Competency Based Questions and Answers in Ophthalmology

for Third MBBS Professional Examination

Compiled and designed as per
Competency Based Undergraduate Curriculum for the Indian Medical Graduate
prescribed by Medical Council of India

- 64 Long Essays
- 343 Short Essays
- 385 Short Answers
- 305 MCQs

Madhurima K Nayak
Sushrutha Academy

CBSPD *Dedicated to Education*
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Madhurima K Nayak MS, DNB
Consultant Ophthalmologist
Yenepoya Specialty Hospital
Mangaluru, Karnataka

Sushrutha Academy
Bengaluru, Karnataka

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Corporate Office: 204 FIE, Industrial Area, Patparganj, New Delhi-110092; Ph: +91-11-49344934; Fax: +91-11-49344935; Website: www.cbspd.com; www.eduport-global.com; E-mail: eresources@cbspd.com; marketing@eduport-global.com

Head Office: CBS PLAZA, 4819/XI Prahlad Street, 24 Ansari Road, Daryaganj, New Delhi-110002, India.; Ph: +91-11-23289259, 23266861, 23266867; Fax: 011-23243014; Website: www.cbspd.com; E-mail: publishing@cbspd.com; eduportglobal@gmail.com.

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to

Mrs Mita A Nayak
My Best Friend and Mother
for her ever-inspiring role in my life

Preface

Competency Based Questions and Answers in Ophthalmology is my first book as an author. As a student, I used to wonder, if I would write a book in the future, how would my preface be? Believe me, I am still wondering.

As a medical student, you first learn from your patients, and so, respect them. Next, you learn from your teachers and professors, friends and peers, from journals and conferences, and lastly from textbooks. I believe in conventional learning through standard textbooks and reference books.

My purpose in writing this book is to make ophthalmology an easy and digestible subject for the students who think it as a very difficult and dull subject. And also for those of you who love ophthalmology, well, welcome aboard!! With the introduction of CBME, learning ophthalmology is more methodical and clearer. With this question bank, I am sure, you will be able to understand the type of questions that can be expected in your professional examinations and the way of structuring your answer for any question. Though there are 46 learning competencies in the new curriculum, I have taken the liberty to add a few miscellaneous topics under each chapter to include those topics which have been omitted in the present curriculum. These might not be important for your professional examinations, but they certainly help you to understand the subject.

By no means, this book is a substitute for the standard textbooks and clinical learning. It serves as a last-minute revision book to cope with the exam tension.

Also, there are multiple-choice questions at the end of this book, some of which are case-based and image-based. These may help you prepare for the upcoming examinations.

There is no substitute for hard work, and you learn better when you love the subject.

Wish you a happy learning experience and best wishes!

Madhurima K Nayak

Acknowledgements

I would like to begin my acknowledgments by thanking the Almighty, *Paramatman*, who is my strength, my inspiration, and my knowledge.

I am deeply indebted to Sushrutha Academy for this opportunity and encouragement.

I dedicate this book to my parents, Mita and Arun Nayak, who have always supported me in all my endeavors.

My gratitude to my elder brother, Abhinav, who inspired me to take up ophthalmology and even gifted me a laptop to begin writing my book.

I must thank my ophthalmology teachers, Dr Gurudutt Kamath, Dr Manjunath Kamath, Dr Sumana Kamath, Dr Shobha Pai, Dr Ajay Kamath, Dr Anupama Karanth, and Dr Rajesh Nayak, for making me fall in love with ophthalmology and harboring a passion for academics.

My love and gratitude to my five-year-old son, Kartikeya, who is very eager to read my book and always let me do my writing work. My heartfelt thanks to my husband, Dr Kashinath, for his all-around support that helped me author this book.

I would like to extend my gratitude to Dr Tejaswi HL, for guiding me with the finer aspects of book writing.

I sincerely thank Ms Kavya Kamath for helping me with the typing work.

I would like to acknowledge all those who have been involved in the preparation of this book, especially Mr SK Jain (Chairman and Managing Director), Mr Varun Jain (Director), Mr YN Arjuna (Sr. Vice President—Publishing, Editorial and Publicity), Mrs Ritu Chawla (GM Production), Mr Dinesh Chandra Gupta (Senior editor), Mr Parmod Kumar (DTP operator) and Mr Rohan Prasad (Graphic designer) of CBS Publishers & Distributors for their all-time support in bringing out this book in record time.

I hereby wish all the readers of the book all the best in their endeavors.

Madhurima K Nayak

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Details of the Number of Questions and MCQs Included as per the Competency

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7	OP2.2	Demonstrate the symptoms and clinical signs of conditions enumerated in OP2.1	—	2	3	5
8	OP2.3	Demonstrate under supervision clinical procedures performed in the lid including: Bell's phenomenon, assessment of entropion/ectropion, perform the regurgitation test of lacrimal sac. Massage technique in cong. dacryocystitis, and trichiatic cilia removal by epilation	—	—	3	6
9	OP2.4	Describe the aetiology, clinical presentation. Discuss the complications and management of orbital cellulitis	1	1	7	5

S. No.	*Competency No.*	*Competency Details*	*Long Essays*	*Short Essays*	*Short Answers*	*MCQs*
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11	OP2.6	Enumerate the causes and describe the differentiating features, and clinical features and management of proptosis	2	5	5	6
12	OP2.7	Classify the various types of orbital tumours. Differentiate the symptoms and signs of the presentation of various types of ocular tumours	—	—	6	3
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15	OP3.2	Demonstrate document and present the correct method of examination of a "red eye" including vision assessment, corneal lustre, pupil abnormality, ciliary tenderness	—	1	5	3
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S. No.	*Competency No.*	*Competency Details*	*Long Essays*	*Short Essays*	*Short Answers*	*MCQs*
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S. No.	*Competency No.*	*Competency Details*	*Long Essays*	*Short Essays*	*Short Answers*	*MCQs*
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39	OP6.5	Describe and discuss the angle of the anterior chamber and its clinical correlates	3	2	2	5
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S. No.	*Competency No.*	*Competency Details*	*Long Essays*	*Short Essays*	*Short Answers*	*MCQs*
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47	OP7.3	Demonstrate the correct technique of ocular examination in a patient with a cataract	—	2	2	5
48	OP7.4	Enumerate the types of cataract surgery and describe the steps, intra-operative and post-operative complications of extracapsular cataract extraction surgery	2	15	10	5
49	OP7.5	To participate in the team for cataract surgery	—	—	4	5
50	OP7.6	Administer informed consent and counsel patients for cataract surgery in a simulated environment	—	1	2	5
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S. No.	*Competency No.*	*Competency Details*	*Long Essays*	*Short Essays*	*Short Answers*	*MCQs*
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58	OP9.3	Describe the role of refractive error correction in a patient with headache and enumerate the indications for referral	—	2	4	4
59	OP9.4	Enumerate, describe and discuss the causes of avoidable blindness and the National Programs for Control of Blindness (including vision 2020)	1	2	—	4
60	OP9.5	Describe the evaluation and enumerate the steps involved in the stabilisation, initial management and indication for referral in a patient with ocular injury	4	18	12	7
61		Miscellaneous	—	2	4	—
		Total content	**64**	**343**	**385**	**305**
		Total number of Fill in the Blanks: 217				

CHAPTER

1

Visual Acuity Assessment

COMPETENCY

OP1.1 DESCRIBE THE PHYSIOLOGY OF VISION

LONG ESSAY

1. A 22-year-old gentleman was tested for color vision as a requisite for his driver's license. He could not identify some plates in a standardized testing format.

A. Define color sense. Name the photoreceptors involved in color vision. (1+1 marks)

B. Explain the theories of color vision. (3 marks)

C. Enumerate 4 tests used to test color vision. (2 marks)

D. Compare and contrast congenital and acquired color blindness. (3 marks)

A. Color Sense/Color Vision and Photoreceptors

Color Sense/Color Vision

It is the ability of the eye to differentiate various colors as excited by light of varying wavelengths.

Photoreceptors Involved in Color Vision

Cones

B. Theories of Color Vision

1. **Young Helmholtz trichromatic theory**
   - According to this theory, there are three types of color receptors—one for each primary color
     - i. Cones containing "erythrolabe" pigment are most sensitive to absorb the red color
     - ii. Cones containing "çhlorolabe" pigment are most sensitive to absorb the green color
     - iii. Cones containing "cyanolabe" pigment are most sensitive to absorb the blue color
   - Other colors are perceived due to a varying combination of stimulation of cones containing these pigments

2. **The opponent color theory**
   - This theory was proposed by Ewald Hering but has been updated by Hurvich and Jameson
   - According to this theory, one member of the color pair suppresses the appreciation of the other. So, they become mutually exclusive.
     i. Spectrally opponent receptors include red-green and blue-yellow
     ii. Aspectrally opponent receptors include those for black and white
   - Therefore, a color like greenish-red cannot exist
   - This theory explains the concept of after-images and color contrast
3. **Granit's polychromatic theory**
   - This theory believes that the cones have 7 types of receptors—one for VIBGYOR
   - It consists of modulators and dominators. The former is for hue detection and the latter has a wide spectrum for color detection

C. Tests Used for Color Vision

1. Ishihara's pseudoisochromatic charts (most commonly used)
2. Holmgren's wools test
3. Hardy–Rand–Rittler charts (used to detect congenital color blindness)
4. Farnsworth Munsell 100 hue test (most sensitive)

D. Congenital vs Acquired Color Blindness

Feature	Congenital	Acquired
Presentation	Asymptomatic, detected on screening	Maybe symptomatic due to associated ocular pathology
Pattern	Bilateral, symmetrical	It May be unilateral or bilateral Need not always be symmetric
Gender predilection	More in men (as the gene for red-green sensitive cones is present on the long arm of the X chromosome)	No such predilection
Color affected	Red-green is the most commonly encountered defect	Blue-yellow impairment is seen in macular disorders. Red-green impairment is seen in optic nerve diseases
Ocular findings	Visual acuity is usually normal except in rod monochromatism where the visual acuity is around 20/200	Visual acuity may be subnormal as the cause is either due to optic nerve or macular pathology
Fundus findings	Usually normal	It may be abnormal disc or macular findings may be there
Natural history	Stationary and permanent	May progress or improve depending on the pathology
Tests used	Isochromatic charts are most useful (Ishihara and HRR charts)	Farnsworth Munsell 100 hue test and Nagel's anomaloscope are most useful

SHORT ESSAYS

1. Explain phototransduction. (5 marks)

Phototransduction

- It is a process by which light energy is converted into electrical energy in the specialized cells of the retina called photoreceptors—rods and cones

- When light is absorbed by the photoreceptors in the form of photons, the reactions that follow are divided into three steps:
  1. **Rhodopsin bleaching**
     - Rhodopsin is the visual pigment present in the rods. In the dark-adapted state, the opsin is coupled with retinine called 11-*cis*-retinol
     - When light falls on rhodopsin, the 11-*cis*-retinol is converted into *all-trans*-retinal. This is unstable and cannot remain associated with the opsin. This process is called "photodecomposition" (Fig. 1.1.1)
  2. **Rhodopsin regeneration**
     - The 11-*cis*-retinal is generated from the *all-trans*-retinal and combines with vitamin A that is supplied by the blood
     - This process is independent of light
  3. **Electrical changes**
     - The activated rhodopsin (metarhodopsin) is responsible for a cascade of biochemical reactions within the inner and outer segments of the photoreceptors that cause hyperpolarization
     - The inner segment of the photoreceptors passively pumps out sodium ions which are pumped back into the outer segment from the ECF
     - The activated rhodopsin causes a conformational change in opsin that causes the Na^+ channels in the outer segment to close because of which there is hyperpolarization
     - A receptor potential is set up

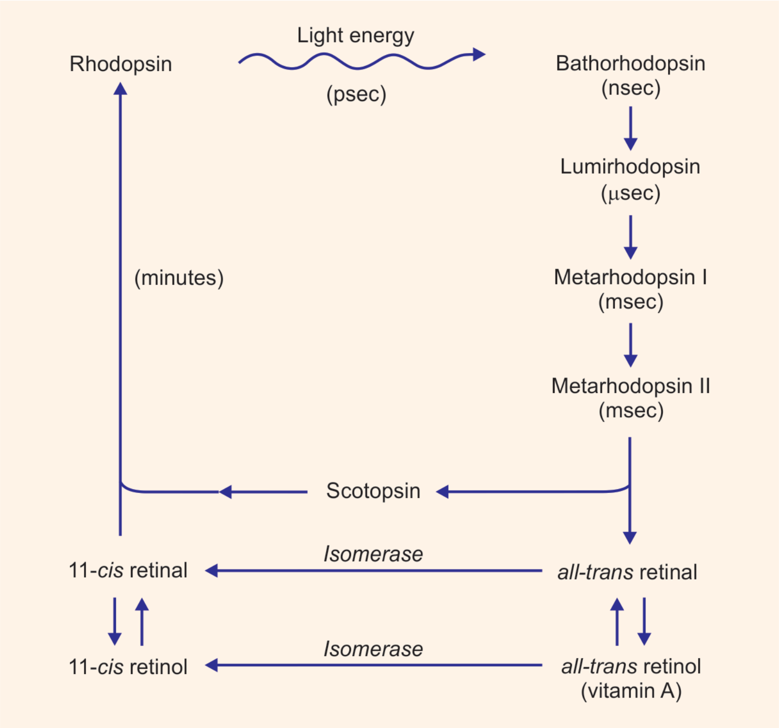

Rhodopsin
Light energy
(psec)
Bathorhodopsin
(nsec)
Lumirhodopsin
(µsec)
Metarhodopsin I
(msec)
Metarhodopsin II
(msec)
(minutes)
Scotopsin
11-*cis* retinal
Isomerase
all-trans retinal
11-*cis* retinol
Isomerase
all-trans retinol
(vitamin A)

Fig. 1.1.1 Photodecompensation

2. Contrast between magnocellular and parvocellular pathways. (5 marks)

These pathways begin in the retinal ganglion cells and run through the entire visual pathway and are responsible for the parallel processing of an image

Feature	Parvocellular pathway	Magnocellular pathway
Diameter	Smaller	Larger
Cells	P cells	M cells
Location	Dominant in the macula	Dominant in the periphery
Layers of lateral geniculate body	3–6	1–2
Speed of conduction	Slower as the diameter is smaller	Faster as the diameter is larger
Color contrast	Yes	No
Spatial frequency	Higher	Lower
Temporal frequency	Lower	Higher
Part of cortex	Project into the primary visual cortex (4Cα of V1)	Project into the primary visual cortex (4 A and 4Cβ of V1)

3. A 14-year-old boy finds it difficult to walk in dim illumination.

A. Name 4 important causes for the same. (2 marks)

B. Explain the dark adaptation curve. (3 marks)

A. Causes of Night Blindness

- This condition is called nyctalopia or night blindness. It means the individual finds it difficult to see in the dark
- Causes are:
  1. Vitamin A deficiency
  2. Retinitis pigmentosa
  3. Miotic pupil
  4. Peripheral cataract

B. Dark Adaptation Curve

- Dark adaptation is the ability of an eye to adapt itself to decreasing illumination
- When an individual moves from a brightly illuminated area into a dimly illuminated region, the time taken for that person to be able to see the objects is called "dark adaptation time". It is around 20–25 minutes

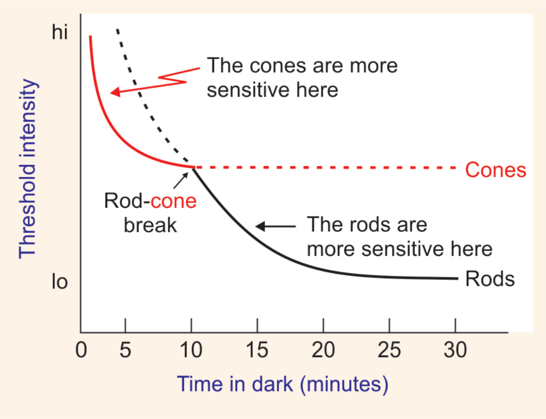


Fig. 1.1.2: Dark adaptation curve

- This curve is plotted by illuminating a certain portion of the retina that has both rods and cones (11 degrees from the fovea) (Fig. 1.1.2)
- There is an early cone adaptation seen as an increase in retinal sensitivity. It is smaller. This point is called the "cone threshold" and is about 5 minutes in duration
- This is followed by a longer, slower, and more sustained, and is formed by regeneration of rhodopsin in rods
- The point of a break between the two is called the 'alpha' point or rod-cone break
- When there is a delay in the dark adaptation time as described in the above-mentioned clinical situations, the curve is shifted higher

4. A predator animal has both eyes facing ahead whereas a prey has eyes on both sides of the face. Explain the advantages and disadvantages of each. Explain the requisites of a binocular singular vision. (2+3 marks)

A Predator has Both Eyes Facing Forward

Advantages

1. There is stereopsis, hence better localization of the object of interest (prey)
2. There is binocular coordination and hence, objects simulate corresponding retinal elements

Disadvantage

The visual field is not as great as when the eyes are on either side of the head.

A Prey has Eyes on Either Side of the Face

Advantage

They have an increased visual field and thus can anticipate attack from a predator more readily.

Disadvantage

It was initially thought that these animals have no depth perception until very recent studies have shown that these animals have an extra temporal fovea which is associated with stereopsis.

Requisites of Development of Binocular Singular Vision

1. Binocular singular vision is a state of simultaneous perception of a single image when light rays emanating from a single object are received by corresponding points of two retinae
2. Fixation
    - The eyes must have their optical axes aligned in such a way that the rays of the object of interest are focused onto the respective foveae
    - The axes of the eyes must be parallel
3. Overlap of visual fields to a large extent is necessary
4. The images formed in each eye should be of comparable sizes, shapes, and colors
5. The retinae must possess physiologically corresponding points
6. The reflex ocular movements must be well developed

5. Explain the grades of a binocular singular vision. (5 marks)

Grades of a Binocular Singular Vision (Fig. 1.1.3)

Conventionally, there are three grades of binocular singular vision and they are measured using a synaptophore

1. **Grade I/simultaneous macular perception**
    - Both eyes can see two dissimilar objects simultaneously
    - When two dissimilar objects are presented to both eyes through a synaptophore, there are fused. For example, when a cage is shown to the right eye and a parrot in the left eye, the binocular system perceives it as a bird in the cage
2. **Grade II/fusion**
    - The brain can fuse two incomplete similar objects to create the image of a single object. It has two components: sensory and motor

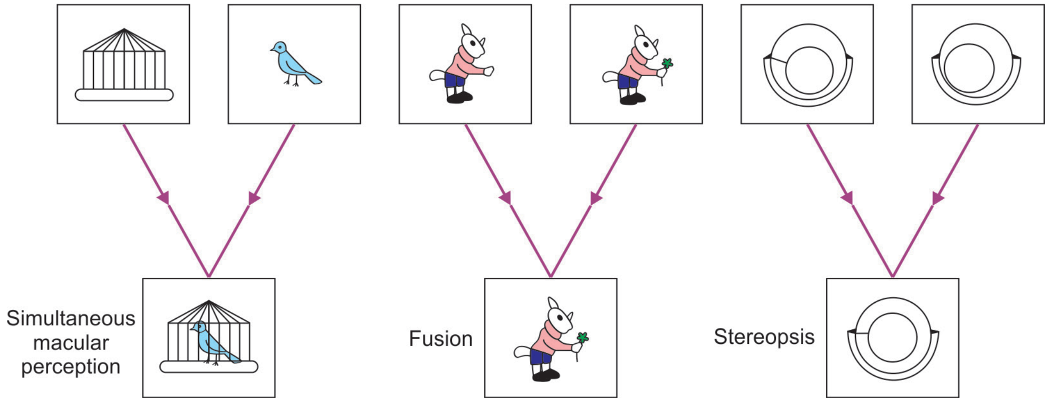


Fig. 1.1.3: Grades of binocular singular vision

- When two partial images are presented to each eye at the same time through a synaptophore, they are seen as a fused image.
- For example, when a rabbit is projected to the right eye and hands holding a bouquet in the left eye, they are seen as a rabbit holding a bouquet

3. **Grade III/stereopsis**
   - It is the most advanced form of binocular vision and it is the ability to perceive the third dimension, i.e. depth perception

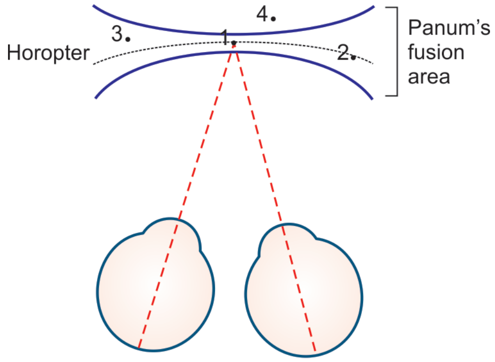
6. Define Panum's fusional area and its role in stereopsis. (1+4 marks)

Panum's Fusional Area

- A horopter is an imaginary circle that joins various points of fixation in the space formed by stimulation of corresponding retinal points in both retinae
- Objects that are placed in front or behind the horopter produce diplopia
- However, a small area straddling this horopter will not lead to the expected diplopia and this space is known as Panum's fusional space (Fig. 1.1.4)
- It is hyperbolic in shape and narrowest at fixation

Role in Stereopsis

- Any point on the horopter does not induce diplopia
- Any point outside the horopter, but within the Panum's fusional space causes the perception of depth or thickness. Thus, this concept of Panum's area helps in understanding stereopsis
- The synergy hypothesis of Panum states that stimulus on one retina can be fused with a range of similar stimuli on points very near the corresponding point on the other retina.
- So, there is fusion with retinal disparity giving rise to the perception of depth



1. Fixation
2. Point on horopter: No retinal disparity
3. Point in Panum's fusion area: Disparity induces stereopsis
4. Point outside Panus's fusion area: Induces diplopia

Fig. 1.1.4 Panum's fusional area

- As the Panum's area is smallest at fixation, fusion ability is the least.
- Therefore, stimuli that lie on either side of the horopter can cause diplopia that the points in the peripheral field of vision

SHORT ANSWERS

1. Anxious parents of a 4-week-old child present to you saying that their baby does not see the mother's face. Is this normal? Explain the development of vision in a child with clear ocular media and normal visual pathways. (1+2 marks)

- Yes. It is normal
  - When a child is born, her visual acuity is roughly 6/60 and is hypermetropic
  - The child is most interested in her close surroundings only, but cannot fixate with both eyes
  - Development of fovea or foveation takes a minimum of 6–8 weeks

Development of Vision in a Child with Clear Ocular Media and Normal Visual Pathways

- For normal visual development, the requisites are adequate light, normally aligned optical axes of both eyes, clear optical media (cornea, lens, and vitreous), and a normal nervous system
- Development of the visual system includes specialization of photoreceptors, maturation of synapses, myelination of the proximal visual pathways, and maturation of the ciliary body
- At birth, the child cannot fixate, and the ocular axis may seem non-parallel
- The first 6 weeks are extremely important as the process of "foveation" takes place
  - At around 6–8 weeks, the child steadily fixes on light or an object of interest held at 8–10 inches
  - At around 12–14 weeks, the child follows when the object is moved
  - At around 5–6 months, stereopsis, color vision, and eye-body coordination develop

2. A couple is planning for a child and wants to know if their child may develop color blindness as the father is color blind since birth. Describe the inheritance, and clinical features of congenital color blindness. (2+1 marks)

Congenital Color Blindness

Inheritance (Fig. 1.1.5)

- The gene for red and green pigments in cones is expressed on the X chromosome
- Congenital color blindness is acquired by X-linked recessive inheritance and is most commonly seen in boys
- If the mother is a carrier, there is a 50% chance of her son acquiring the disease and a 50% chance of her daughter carrying the disease. If the father is affected, all his daughters become carriers

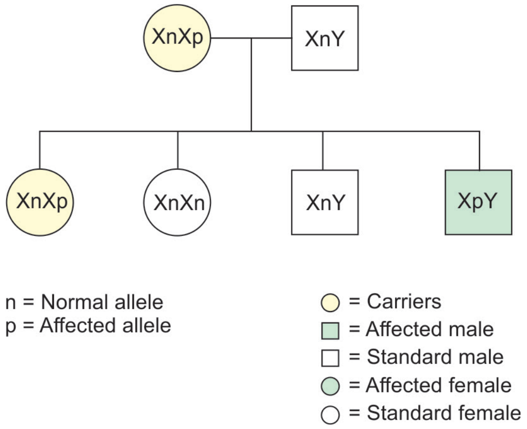


Fig. 1.1.5: Inheritance of congenital color blindness

Clinical Features

- Color-blind individuals appreciate various colors in shades of gray and thus are hardly aware of their defects
- Visual acuity, pupil, and fundus are normal
- Depending on which color they are unable to appreciate, they are categorized as
  - **Protanopia:** Cannot recognize red color
  - **Deuteranopia:** Cannot identify green color
  - **Tritanopia:** Cannot identify blue color

3. Name 3 electrophysiological tests used to test the integrity of the visual system. State their principles. (1+2 marks)

Test	Principle
Visually evoked potential	• It is an objective test that records impulses at the occipital cortex following stimulation of the retina by light • It tests the integrity of the visual system, more so of the macula
Electroretinogram	• The changes in resting potential in the eye upon stimulation by light are recorded through electrodes placed on the cornea
Electrooculogram	• The difference between the potential that exists between the cornea and the posterior aspect of the retina is recorded in a fully dark-adapted and a light-adapted state

4. Enumerate important milestones in the development of visual development. (3 marks)

- At birth: the child cannot fixate, and the ocular axis may seem non-parallel
- The first 6 weeks are extremely important as the process of "foveation" takes place
- At around 6–8 weeks, the child steadily fixes on light or an object of interest held at 8-10 inches
- At around 12–14 weeks, the child follows when the object is moved
- At around 5–6 months, stereopsis, color vision, and eye-body coordination develop

5. Cyclops is a character in Greek mythology who had one eye in the center of the forehead. Explain how, this concept is used in understanding binocular singular vision. (3 marks)

- The concept of the cyclopean eye arises from the concept of corresponding retinal elements
- When corresponding retinal elements of both eyes are stimulated, there is a sensory fusion that takes place
- Corresponding retinal elements create a single visual direction for a particular stimulus
- As a result, a mental impression of the visual direction of both eyes is formed such that an imaginary eye exists between the two eyes in the

forehead where the object is in exact line with the cyclopean eye (Fig. 1.1.6)

- As a result, there is a binocular singular vision and there is no diplopia

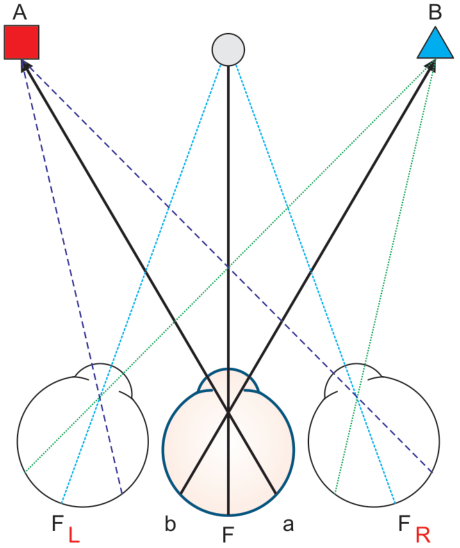


Fig. 1.1.6: Cyclopean eye

6. Explain the physiological basis for electroretinogram. (3 marks)

- Electroretinogram is a recording of the changes of resting potential in the eye that is caused by light
- There exists a potential difference of 1mV between the cornea and the retina and is called the corneo-retinal potential
- When light falls on the retina, there is a change in this potential
- The resultant waveform generated by the action of a flash of light is called the electroretinogram or the ERG
- It is a composite recording of the activity of the Photoreceptors, Muller cells, and bipolar cells through which electrical activity flows
- A typical ERG consists of a negative 'a' wave followed by a large positive 'b' wave
- When light falls on the retina, there is hyperpolarization of the photoreceptors which become more positive when compared to the cornea
- Thus, a negative wave is generated and it represents the activity of the photoreceptors
- The Muller cells in connection with the photoreceptors and release of potassium ions by the photoreceptors cause electrical activity of the Muller cells and a positive wave is generated
- Finally, the 'b' wave is followed by a long positive 'c' which is due to the activity of retinal pigment epithelium (Fig. 1.1.7)

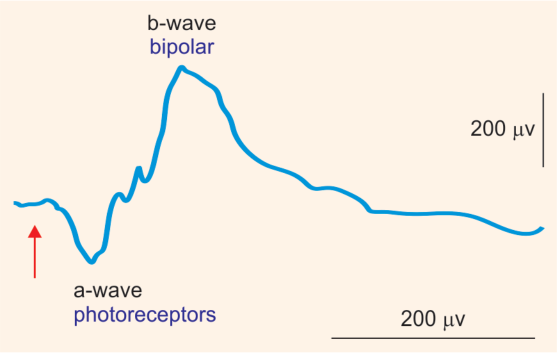
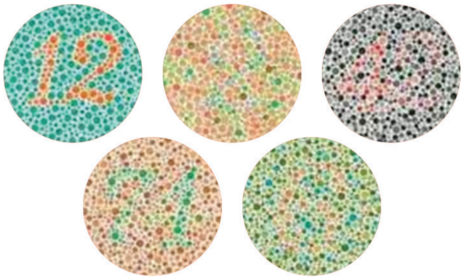


Fig. 1.1.7: Normal ERG

7. A. Identify the test shown in the image. B. State its use. C. Name two causes for acquired color blindness. (1+2=3 marks)



A. The Test Shown in the Picture

- The Ishihara color vision test

B. Use

- It is used to identify and diagnose congenital color blindness

C. Causes of Acquired Color Blindness

1. Optic neuritis
2. Diabetic macular edema

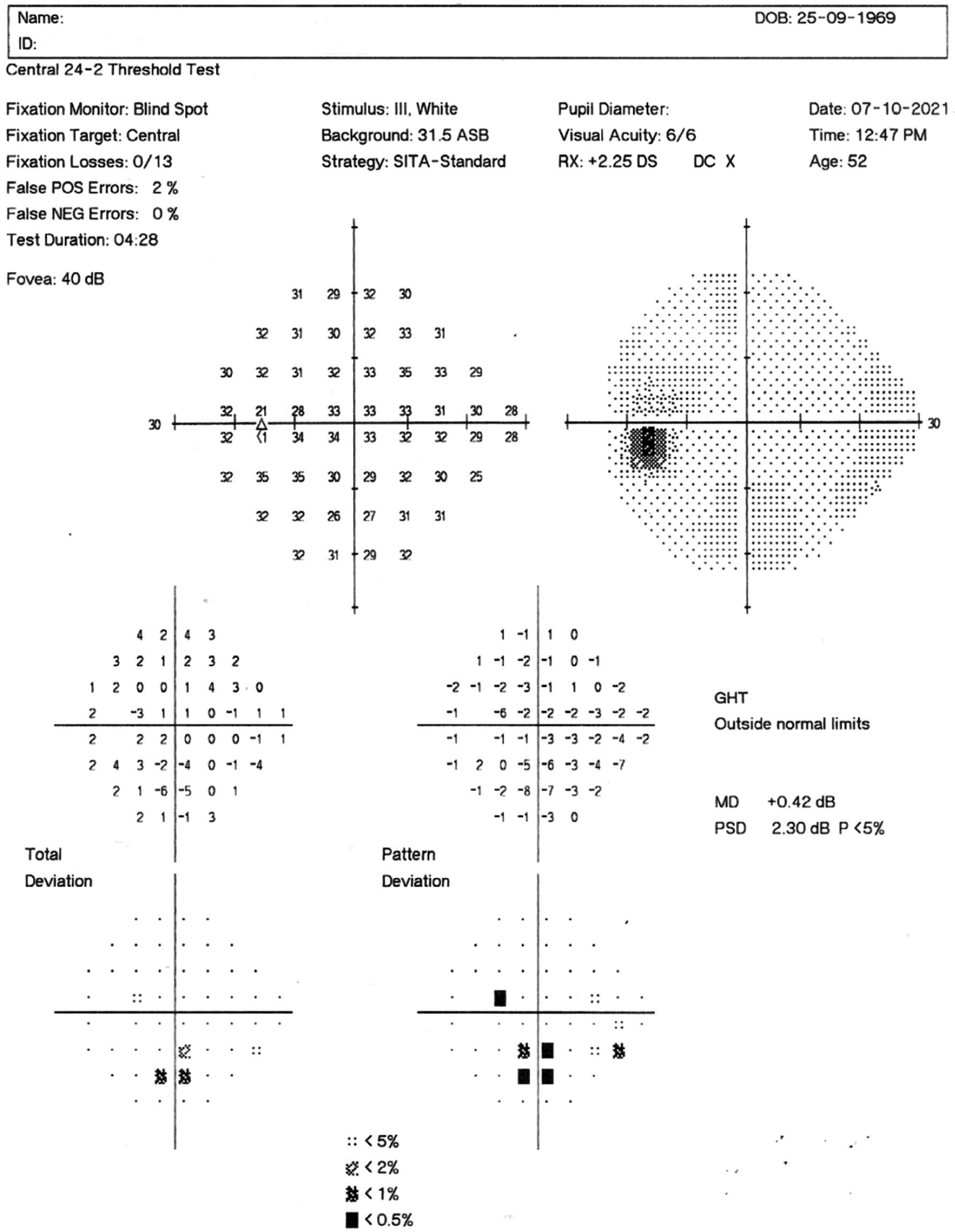
8. A. Identify the test by the report shown in the image.

B. Define a scotoma.

C. Differentiate between a positive and a negative scotoma. (1+1+1 marks)

A. The Image is a Report

- Visual field charting and hence, the test is called perimetry



Name: DOB: 25-09-1969
ID:
Central 24-2 Threshold Test

Fixation Monitor: Blind Spot
Fixation Target: Central
Fixation Losses: 0/13
False POS Errors: 2 %
False NEG Errors: 0 %
Test Duration: 04:28

Fovea: 40 dB

Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA-Standard

Pupil Diameter:
Visual Acuity: 6/6
RX: +2.25 DS DC X

Date: 07-10-2021
Time: 12:47 PM
Age: 52

31 29 32 30
32 31 30 32 33 31
30 32 31 32 33 35 33 29
32 21 28 33 33 33 31 30 28
32 <1 34 34 33 32 32 29 28
32 35 35 30 29 32 30 25
32 32 26 27 31 31
32 31 29 32

4 2 4 3
3 2 1 2 3 2
1 2 0 0 1 4 3 0
2 -3 1 1 0 -1 1 1
2 2 2 0 0 0 -1 1
2 4 3 -2 -4 0 -1 -4
2 1 -6 -5 0 1
2 1 -1 3

Total
Deviation

1 -1 1 0
1 -1 -2 -1 0 -1
-2 -1 -2 -3 -1 1 0 -2
-1 -6 -2 -2 -2 -3 -2 -2
-1 -1 -1 -3 -3 -2 -4 -2
-1 2 0 -5 -6 -3 -4 -7
-1 -2 -8 -7 -3 -2
-1 -1 -3 0

Pattern
Deviation

GHT
Outside normal limits

MD +0.42 dB
PSD 2.30 dB P <5%

:: < 5%
▨ < 2%
▩ < 1%
■ < 0.5%

B. Definition of Scotoma

- A non-seeing area within the visual field

C. Differentiation between a Positive and a Negative Scotoma

Feature	Positive scotoma	Negative scotoma
Definition	Perception of a non-seeing area within the visual field	Non-perception of a non-seeing area within the visual field
Symptoms	A black spot is seen by the patient	Usually asymptomatic
Causes	Neurological	Macular disorders like central serous retinopathy

9. There is a genetic disorder that predominantly affects the cones of the retina. State 2 components of vision that are affected due to cone dysfunction. Give an example of rod dysfunction. (2+1 marks)

Components of Vision that are Affected Due to Cone Dysfunction

- Cones are responsible for day vision and color vision
- So, the patient will experience hemeralopia (day-blindness) and dyschromatopsia (color blindness).
- The central vision is affected as most of the cones are concentrated in the macula which represents the center of the visual field

Example of Rod Dysfunction

Retinitis pigmentosa or pigmentary retinopathy

COMPETENCY

OP1.2 DEFINE, CLASSIFY AND DESCRIBE THE TYPES AND METHODS OF CORRECTING REFRACTIVE ERRORS

LONG ESSAYS

1. A defense personnel undergoes eye testing for vision and his visual acuity was found to be 6/6 in both eyes.

A. Define emmetropia and ametropia. (2 marks)

B. Describe the different types of refractive errors and their clinical features. (6 marks)

C. Enumerate the methods of treating each refractive error. (2 marks)

A. Emmetropia and Ametropia

Emmetropia

It is the state of the eye when parallel rays of light traveling from infinity are focused at a point on the retina with accommodation being at rest.

Ametropia

- It is a state of the eye when parallel rays of light traveling from infinity are not focused at a point on the retina with accommodation being at rest
- It is the presence of a refractive error

B. Different Types of Refractive Errors

1. Myopia

- It is a type of ametropia where light rays traveling from infinity are focused onto a point anterior to the retina. (Fig. 1.2.1)
- As a result, the image is formed in front of the retina
- Myopia is due to increased curvature of the cornea, increased curvature of the anterior surface of the lens, increased axial length of the eyeball, anteriorly displaced lens, or increased sclerosis of the lens
- Clinical features
  - The patients are usually in their school-going age
  - They present with an inability to see distant objects clearly and hence myopia is also known as "nearsightedness" in layman's terms
  - On examination, the eyeballs appear large, deep anterior chamber and fundus may be normal in lower degrees of myopia
  - In degenerative myopia, there are retinochoroidal degenerative features seen on fundoscopy
- Diagnosis by
  - Retinoscopy

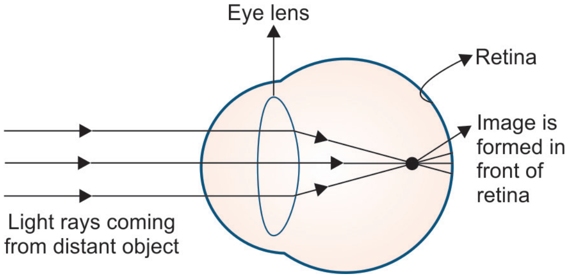

Eye lens
Retina
Image is formed in front of retina
Light rays coming from distant object

Fig. 1.2.1: Mechanism of myopia

2. Hypermetropia

- It is a type of refractive error where the light rays traveling from infinity are focused onto a point posterior to the retina (Fig. 1.2.2)
- As a result, the image is formed behind the retina
- Hypermetropia can result due to flat cornea, flat anterior surface of the lens, posteriorly displaced lens, short axial length, or sclerosis of the cortex
- Clinical features
  - The individuals usually complain of headaches in mild degrees of hypermetropia
  - The headache increases with near work and the end of the day
  - Asthenopia or tiredness of eyes and difficulty in seeing nearby objects or letters is a classical symptom
  - On examination, the eye may appear smaller, with a shallow anterior chamber and fundus has an appearance of 'silk shot retina' that is, increased reflexes on the retina with 'pseudo-papillitis' due to small optic nerve head may simulate disc edema
- Diagnosis by
  - Retinoscopy

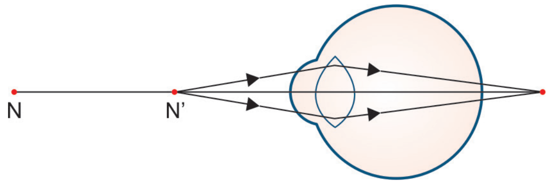


Fig. 1.2.2: Mechanism of hypermetropia

3. Astigmatism

- It is a type of ametropia where incident parallel rays of light traveling from infinity cannot be brought to focus on to one point on the retina with any spherical lens, rather focus onto multiple points along a line (Fig. 1.2.3)
- Astigmatism is most commonly due to irregular cornea, followed by lenticular astigmatism that can be seen in mild cases of subluxation and lenticonus.

- Clinical features
    - The individuals usually complain of headaches and may complain of distorted vision
    - Asthenopia or tiredness of eyes and difficulty in seeing nearby objects or letters is a classical symptom
    - On examination, the eye appears normal in small degrees of astigmatism
- Corneal scars may be present that cause larger degree of astigmatism

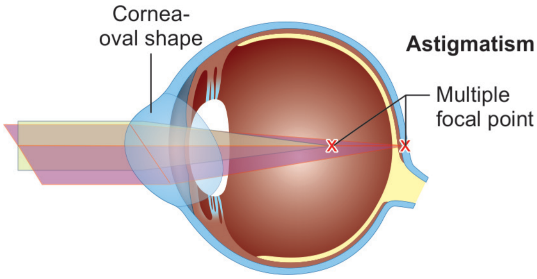


Fig. 1.2.3: Mechanism of astigmatism

C. Methods of Treating Each Refractive Error

1. **Myopia**
    - Concave lenses in the form of spectacles and contact lenses
    - Refractive surgeries like photorefractive keratectomy, LASIK, phakic IOL, and refractive lens exchange
2. **Hypermetropia**
    - Convex lenses in the form of spectacles and contact lenses
    - Refractive surgeries like hyperopic LASIK, conductive keratoplasty
3. **Astigmatism**
    - Cylindrical lenses in the form of spectacles and contact lens. Rigid contact lenses and toric contact lenses are preferred in higher degrees of astigmatism
    - Refractive surgeries include arcuate keratotomy and astigmatic LASIK

2. An 11-year-old girl presents with difficulty in seeing letters written on the blackboard.

A. Define myopia. **(1 marks)**

B. Classify myopia. **(3 marks)**

C. Enumerate two secondary causes of myopia. **(2 marks)**

D. Explain the principles of treatment and correction of myopia. **(4 marks)**

A. Myopia

A type of refractive error in which incident rays traveling from infinity are brought to focus on a point in front of the retina.

B. Classification of Myopia

Based on the clinical presentation

1. Simple myopia/school myopia: presents with a mild degree of myopia, usually not more than –6D
2. Developmental myopia: usually seen in infants born with a congenitally long eyeball and around –10D of non-progressive myopia
3. Degenerative myopia: a type of progressive myopia where there is degeneration of the choroid and the retina with an increasing axial length of the eyeball

C. Secondary Causes of Myopia

Cornea	Keratoconus
Lens	Nuclear sclerosis of the lens
	Hyperglycemia causes increased refractive index of the lens

D. Principles of Treatment

Optical Treatment

- In myopia, the converging power of the eye as an optical system is higher than normal, which is more than +60D
- A concave lens is used to diverge the converging lens so that the rays are made to converge onto the surface of the retina (Fig. 1.2.4)
- The concave lens can be used in the form of spectacles or contact lenses

Advantages of Spectacles

1. Easy to wear
2. Reversible

Disadvantages of Spectacles

1. May restrict the field of vision
2. Minification

Advantages of Contact Lenses

1. Placed over the cornea, hence more physiological
2. Minimal minification
3. Useful in unilateral high myopia

Disadvantages of Contact Lenses

1. Handling difficulty
2. Contact lens associated problems

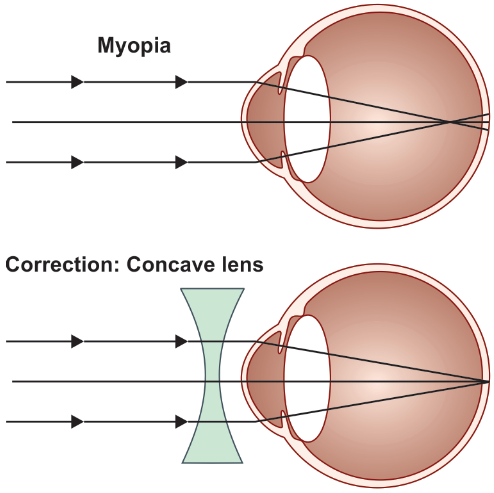


Fig. 1.2.4: Effect of concave lens in myopia

Surgical Correction for Myopia

- Corneal refractive procedures are based on the flattening of the cornea by various keratorefractive procedures or by lenticular procedures
- Flattening can be caused by incisions placed by using blades or using excimer LASER
- Lenticular procedures include refractive lens exchange and phakic IOL insertion

3. A motorist sustained an injury to the cornea following he underwent repair. He complains of distorted vision.

A. Define astigmatism and explain its types.
B. Draw a neat and labeled diagram of the Sturm's conoid.
C. Explain the clinical features and methods of correction of astigmatism.
D. Give two causes for distorted image formation. (3+3+3+1 marks)

A. Astigmatism

Definition

- It is a type of ametropia in which light rays incident on the eye do not get focused onto a point on the retina, instead get focused along a line
- This happens due to the difference in the power between two major meridia of the refracting surfaces

Types

1. **Regular astigmatism**

- In this type, the refractive power of the eye changes regularly from one meridian to another
- It is further divided into four types
    - i. With the rule astigmatism (vertical meridian more curved than horizontal)
    - ii. Against the rule astigmatism (horizontal meridian more curved than the vertical)
    - iii. Oblique astigmatism (the two principal meridia are not vertical and horizontal, but perpendicular to each other
    - iv. Bioblique astigmatism (the two meridia are neither vertical nor horizontal, also not perpendicular to each other)

2. **Irregular astigmatism**

- In this type, the refractive power of the eye changes irregularly from one meridian to another

B. Labeled diagram of the Sturm's Conoid

- Sturm's conoid (Fig. 1.2.5) is a pictorial representation of refraction at an astigmatic surface where the curvature of the horizontal meridian differs from the vertical meridian
- Various positions represent different clinical types of astigmatism:
    - A: Compound hypermetropic astigmatism
    - B: Simple hypermetropic astigmatism
    - C, D, and E: Mixed astigmatism
    - D: Circle of least diffusion
    - F: Simple myopic astigmatism
    - G: Compound myopic astigmatism

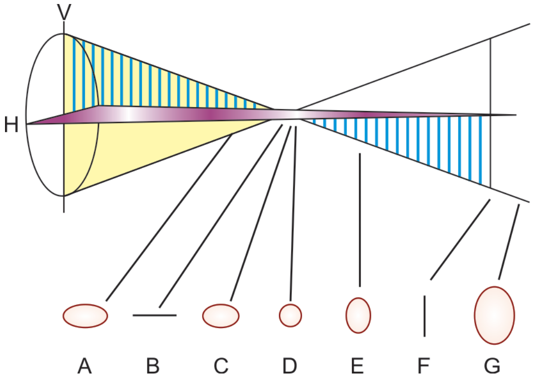


Fig. 1.2.5: Sturm's conoid

C. Astigmatism

Clinical Features

- Astigmatism causes asthenopia and headache more commonly than visual disturbances
- Blurred vision for near or distance depending on the type of astigmatism (hypermetropic or myopic)
- Patients may rarely perceive distorted shapes of objects
- Signs: Cornea may show scars that are responsible for astigmatism. The fundus may show an oblique disc
- Diagnosis is established by retinoscopy when two different powers are needed to neutralize two meridia. Autorefractometry, astigmatic fan, and Jackson's cross-cylinder can be used to confirm the power before prescribing

Methods of Correction

1. Spherocylindrical or cylindrical lenses are the most used form of treatment
2. Soft contact lenses with spherical correction can be useful in powers less than 0.75D
3. Rigid lenses are useful in slightly higher-powered lenses (2–3D)
4. Toric contact lenses are needed in higher powers
5. Astigmatic LASIK, arcuate keratotomy are corneal procedures

D. Causes for Distorted Image Formation

1. Astigmatism
2. Macular disorders

4. A 42-year-old tailor has trouble threading the needle of very recent onset. He never had any visual problems until now.

A. State the most probable diagnosis.	(1 mark)
B. Enumerate the presenting features.	(2 marks)
C. Explain the mechanism of accommodation.	(4 marks)
D. Enumerate the methods of correcting this condition.	(3 marks)

A. Most probable diagnosis

Presbyopia

B. Presenting Features of Presbyopia

- The tiredness of eyes or asthenopia
- Failing vision for near or blurred vision for proximal objects
- Headache, which increases more towards the day
- Intermittent diplopia due to a disturbed relationship between accommodation and convergence may be experienced by some

C. Mechanism of Accommodation

- The most accepted theory for accommodation is by Helmholtz. It is also called the relaxation theory (Fig. 1.2.6)
- According to this, the lens capsule is held taut under tension circumferentially by ciliary zonules that arise from the ciliary ring
- In an unaccommodated state, the lens is kept compressed by the tense zonules and the relaxed ring
- Following stimulation for accommodation, there is a contraction of the ciliary ring leading to shortening and relaxation of the zonules. As a result, the lens capsule relaxes
- This is followed by bulging of the anterior surface of the lens that increases the curvature and decreases the radius of curvature
- As a result, the converging capacity of the refracting surface increases
- A second theory is by Tscherning, according to which contraction of the ciliary ring, leads to contraction of zonules and tightening of the capsule at the equator. As a result, the capsule at the poles of the lens becomes lax and the anterior surface bulges forward

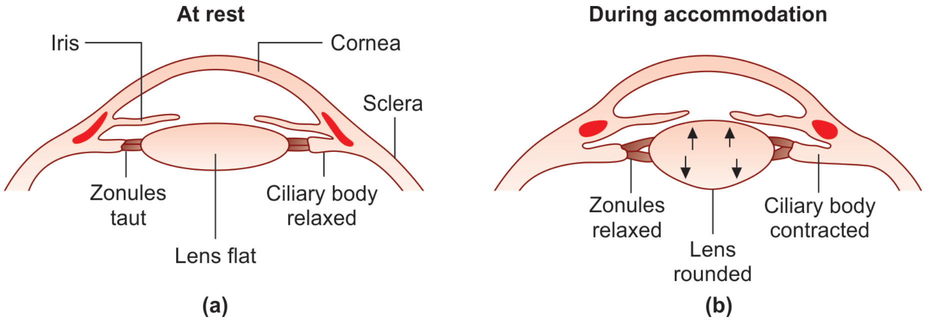


Fig. 1.2.6: Mechanism of accommodation

- This theory was further extended by Schachar. According to this, equatorial tension caused by the tension in the zonules leads to equatorial displacement of the lens and the central part of the lens bulges forward (Fig. 1.2.7)

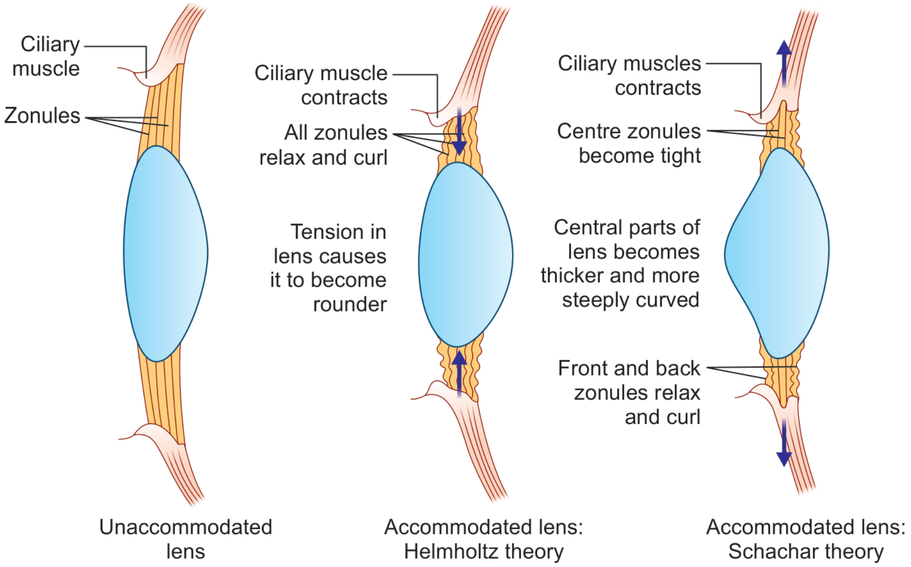

Ciliary muscle
Zonules
Ciliary muscle contracts
All zonules relax and curl
Tension in lens causes it to become rounder
Ciliary muscles contracts
Centre zonules become tight
Central parts of lens becomes thicker and more steeply curved
Front and back zonules relax and curl
Unaccommodated lens
Accommodated lens: Helmholtz theory
Accommodated lens: Schachar theory

Fig. 1.2.7: Comparison between Helmholtz and Schachar theory

D. Methods of Correction

1. Optical Correction

- It is the simplest form of correction
- It is done using convex lenses in the form of spectacles or contact lenses
- The power of the glass is based on the need of the patient, his occupation, working distance, and most importantly preexisting refractive error
- It may range from +1.0D to +3.0D sphere
- The presbyopic correction can be provided in the form of monofocals, bifocals, or progressive glasses

2. Refractive Surgeries

- They are becoming more popular
- Monovision conductive keratoplasty
- Monovision LASIK
- Bifocal LASIK
- Presbyopic multifocal LASIK
- Corneal inlays
- Multifocal accommodating IOLs
- Monovision with IOLs
- Scleral based surgeries (Anterior ciliary sclerotomy, scleral expansion surgery)

SHORT ESSAYS

1. Enlist the components of hypermetropia. State the clinical importance of these components. (4+1 marks)

- Hypermetropia is a type of refractive error in which parallel rays traveling from infinity are brought to a point focus behind the retina
- It is also called 'long-sightedness in layman terms a poorer vision for near

Components of Hypermetropia

1. **Total hypermetropia:** Total hypermetropia after complete cycloplegia
2. **Manifest hypermetropia:** The amount of hypermetropia uncorrected by ciliary tone
3. **Latent hypermetropia:** The amount of hypermetropia that is corrected by inherent ciliary tone
4. **Facultative hypermetropia:** The amount of hypermetropia corrected by voluntary ciliary effort
5. **Absolute hypermetropia:** The amount of hypermetropia that remains uncorrected after accommodating effort

Clinical Importance

While providing an optical correction for hypermetropia, the value of manifest refraction + one-third of the latent refraction should be prescribed (Donder' law).

2. Describe the clinical features of hypermetropia. (5 marks)

Clinical Features of Hypermetropia

Symptoms

- Aesthenopic symptoms such as brow ache, headache, heaviness of eyes, and watering occur more commonly than with myopia as these patients have to use more of their accommodation to get a clear image of nearby objects. These are seen more towards the end of the day
- Recurrent styes, chalazia, and blepharitis. This is probably due to excessive rubbing of eyes due to eye strain
- High uncorrected hypermetropia in childhood can result in a convergent squint
- Amblyopia is more common in hypermetropia as younger children have more interest in nearby objects and when they are unable to do the same, develop amblyopia

Signs

- Short eyeballs, shallow anterior chamber
- The fundus may reveal a bright reflex simulating watered silk, also called shot-silk retina. The optic disc may seem crowded and mimic papillitis and thus the term "pseudopapillitis"
- Diagnosis is established by retinoscopy

3. Describe the features of pathological myopia. (5 marks)

Pathological Myopia/Degenerative Myopia

- Myopia associated with other pathologies of the eye especially the retina and choroid and is genetic in origin
- It is degenerative and progressive
- Usually begins around 5–10 years to progress up to the third decade

- Usually present with rapid fall in far vision which is uncorrectable with refractive correction
- They may also have a lot of floaters due to vitreous degeneration
- The power may go as high as –20 to –25D

Clinical Signs (Fig. 1.2.8)

- Long eyeball evidenced by long axial length around 28–33 mm
- Deep anterior chamber
- May be associated with a posterior subcapsular cataract
- Vitreous degeneration, posterior vitreous detachment
- Extensive retinal thinning and degeneration, peripheral cystoid degeneration
- Large and pale appearing disc with temporal crescent or peripapillary atrophy
- Nasal supertraction of the retina over the disc
- Prominent choroidal vessels due to thinning of the overlying retina
- Lacquer cracks (linear cracks in Bruch's membrane)
- Posterior staphyloma
- Foster Fuchs spot (sub-retinal bleed due to rupture of choroidal vessels at fovea) and myopic choroidal neovascular membrane
- Can be complicated with retinal detachment and vitreous hemorrhage

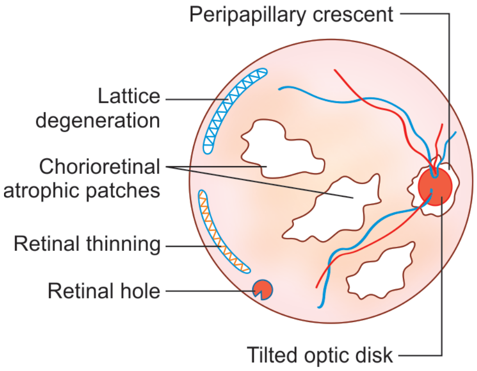


Fig. 1.2.8: Fundus features of degenerative myopia

4. Describe the etiology and clinical features of school myopia. (2+3 marks)

School Myopia

- School myopia or simple myopia is a common form of myopia where the power of the eye usually does not exceed –6D
- It is considered physiological as it occurs with the sudden increase in growth of the eyeball associated with puberty

Etiology

- Axial type:
  - Increase in the axial length of the eyeball causes the image to fall anterior to the retina. 1 mm increase in length of the eyeball causes myopia of roughly 3D
  - It is proposed to be associated with precautious neurological development
- Curvatural type: It is due to underdeveloped eyeball
- Genetics: Myopia is supposed to have a genetic inheritance
- Excessive near work: Myopia progresses more in children who do more near work, have less outdoor activity

Clinical Features

Symptoms

- Diminished vision for distance is usually the main complaint
- Asthenopia and headache may be there with smaller refractive errors
- In younger children, the parents may complain of the child viewing the television from a closer distance, failing to attain milestones, and disinterested in outdoor activities

Signs

- The eyeballs appear long and prominent
- The anterior chamber is deep
- The fundus is usually normal, but some may have a temporal crescent
- Some even may have peripheral retinal degenerations and atrophic holes

5. Define anisometropia? Explain its effects on a binocular singular vision.

(1+4 marks)

Anisometropia

Definition

- It is a condition in which both eyes have different refractive powers
- Clinical definition
  - A difference of more than 2.5D is taken as anisometropia, as up to 2.5D can be tolerated by the cortex
  - Up to 4D can be tolerated based on individuals, more than 4D cannot be tolerated

Effects on Binocular Singular Vision

Anisometropia can cause suppression, amblyopia, or loss of binocular function.

1. **Suppression and amblyopia**
   - Individuals with anisometropia described beyond the above-mentioned degrees cause diplopia or confusion and in a long-term can cause anisometropic amblyopia
   - The mechanism by which amblyopia develops is suppression
   - Even lower degrees of anisometropia can cause a decline in binocularity which is evident on the Worth four dot test
2. **Loss of binocular function**
   - There is a loss of stereoacuity beyond 1 Diopter difference in the sphere
   - The probable mechanism by which stereoacuity is affected is by foveal suppression
   - With an anisometropia of 1D, a clinically acceptable level of stereopsis can be maintained. But with an anisometropia of 4D, only 20% of individuals can maintain a moderate amount of stereopsis
   - Also, the amount of decline in binocularity is more in spherical anisometropia
   - In cases of mixed anisometropia, there is an alternate vision
   - For example, if one eye is moderately hypermetropic and the other eye is myopic, amblyopia does not develop. But there is no binocularity
   - Due to the aforementioned reasons, anisometropia should be appropriately corrected in the pediatric age group

6. Define accommodation and describe the anomalies of accommodation.

(1+4 marks)

Accommodation

Definition

Defined as a process by which divergent rays coming from a near object are converged at a point on the retina with the help of the ciliary body, zonules, lens, and the pupillary aperture.

Anomalies

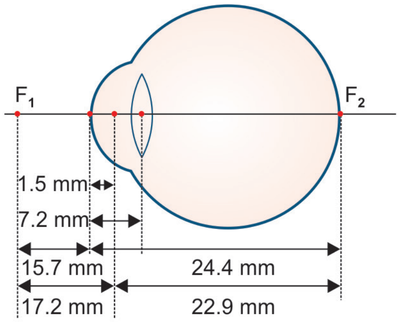
1. **Presbyopia**
    - It is an age-related decrease in accommodating power of the ciliary body and the lens, resulting in an inability to focus on the nearby objects
2. **Insufficiency of accommodation**
    - It is a condition in which accommodation is less than the age-matched normal population. They present with asthenopia and blurred vision for near. It is not ametropia. It is due to a decrease in the amplitude of accommodation
3. **Paralysis of accommodation**
    - It is total paralysis of accommodation
    - Causes can be drug-induced, third nerve palsy, internal ophthalmoplegia due to ciliary ganglionitis
4. **Spasm of accommodation**
    - When the accommodation is abnormally excessive, it is called a spasm of accommodation
    - They usually present with browaches and aesthenopic symptoms
    - They can have blurred vision due to induced myopia
    - Diagnosis is established by atropine retinoscopy
    - Treatment is in the form of cycloplegia with atropine and correction of associated refractive error
    - Avoidance of near work is advised

7. Explain the optics of aphakia. (5 marks)

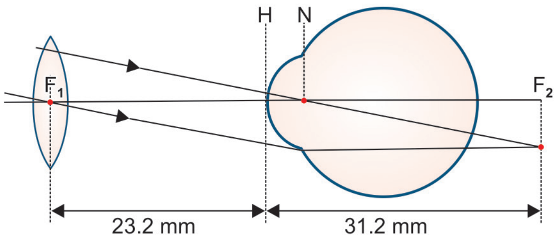
- Aphakia is an absence of a crystalline lens
- The lens contributes to around 18–20D of the total 60D of the eye

Changes in Cardinal Points of the Eye (Fig. 1.2.9)

- The total power of the eye is reduced to around 40–45D. the eye becomes extremely hypermetropic
- The anterior focal point becomes 23.2 mm in front of the cornea and the posterior focal point is shifted to 31 mm behind the cornea
- The two principal points are at the anterior surface of the cornea. The nodal points are close to each other and about 7.75 mm behind the cornea
- An aphakic eye has very poor vision unless the eye is highly myopic. Also, there is against the rule of astigmatism
- There is a loss of accommodation



Reduced eye



Aphakic eye

Fig. 1.2.9: Optics of aphakia

8. Describe various methods of correcting aphakia with the pros and cons of each type. (5 marks)

Methods of Aphakia Correcting

Method	Pros	Cons
• Spectacles • Using highly plus lenses to correct aphakia was the commonest method used in the past • Around +10D power, glasses are used	• Low cost • Easy handling	• Heavy • Magnification up to 30%, can cause diplopia if unilateral • Chromatic and spherical aberration. Spherical aberration causes the pincushion effect • Decreased field of vision • Jack in the box phenomenon • Cosmetically not acceptable
• Contact lenses	• There is no magnification. • No spherical, chromatic aberrations • The field of vision is bigger • No roving ring scotoma	• Difficult to handle as vision is very low • Cost and contact lens-related issues
Surgical correction 1. The PC IOL can be placed in the capsular bag if it is present. 2. Or iris-claw lenses can be used. 3. ACIOL (anterior chamber IOL) which is angle supported was a good option, now they are slowly becoming obsolete. 4. Scleral fixated lenses are also a good option	• More physiological	• Need for a second surgery • Risk of intraoperative procedures • Further decline in endothelial health (especially in ACIOL use)
• Refractive corneal procedures: keratophakia and epikeratophakia (obsolete) • Hyperopic LASIK can be tried	• No intraocular manipulation • Can correct astigmatism	• Cost and availability

9. Compare various types of contact lenses. (5 marks)

Types of Contact Lenses

- Depending on the nature of the material used, contact lenses are divided into three types

Feature	Hard lenses	Rigid gas permeable lenses	Soft lenses
Material	Made of PMMA	Silicone acrylate	HEMA
Special properties		Permeable to oxygen	1–2 mm larger than the corneal diameter
Optical quality	High	High	Lesser
Durability	Durable and cost effective	Durable and cost effective	Expensive

Contd.

Feature	Hard lenses	Rigid gas permeable lenses	Soft lenses
Disadvantages	Impermeable to oxygen, so difficult to tolerate, can cause abrasions		Protein deposits are more common, cannot correct astigmatism >2D
Present-day use	Uncommon	A high degree of astigmatism, keratoconus	Commonly used due to comfort

- Depending on the ionic charge and water content, soft contact lenses are grouped into 4 categories

Group 1	Low water content Nonionic polymers
Group 2	High water content Nonionic polymers
Group 3	Low water content Ionic polymers
Group 4	High water content Ionic polymers

SHORT ANSWERS

1. Mr. J went to an ophthalmologist and was given the following prescription.

Eyecare Hospital, Mumbai

Name Mr. 1 Date: 02/04/2021

Age 55 years

Address: Maharashtra

	Right eye			Left eye		
	Sphere	Cylinder	Degrees	Sphere	Cylinder	Degrees
Distance	+2.0D	+0.5D	180	+1.5D	+0.75D	180
Near	+4.5D	+0.5D	180	+4.0D	+0.75D	180

Remarks: Bifocals for constant use

Signature

A. State the type of refractive error. **(2 marks)**

B. Define presbyopia. **(1 mark)**

A. Type of Refractive Error

Compound hypermetropic astigmatism in both eyes (with against the rule astigmatism) along with presbyopia.

B. Definition of Presbyopia

A physiological age-related decrease in the accommodative power of the crystalline lens leading to difficulty perceived by the individual in seeing clearly at proximity.

2. Explain how each component of the eye contributes to the refractive status with appropriate examples. (3 marks)

Ametropia or refractive error may be due to one or more of the following:

1. **Axial length**
   - Cause of ametropia is the abnormal axial length of the eyeball
   - A long eyeball leads to myopia, whereas a short eyeball leads to hypermetropia
   - 1mm of difference in length causes a difference of 3D in refractive error
2. **Curvature of the cornea and the lens**
   - Cause of ametropia is due to increase or decrease in the radius of curvature of the anterior surface of the cornea and lens
   - 1 mm difference in the radius of curvature produces ametropia equivalent to 6D
   - Thus, steep cornea leads to myopia and flattening of cornea causes hypermetropia
3. **Refractive index**
   - Change in the refractive index of cornea, aqueous, lens, or vitreous leads to ametropia
   - An increase in the refractive index of the lens as in senile sclerosis causes a myopic shift, whereas an increase in the refractive index of the cortex causes a hypermetropic shift
4. **Position of the lens**
   - The nodal point in the eye is the posterior pole of the lens
   - Hence any change in the position of the lens produces a refractive error
   - A forward displacement of the lens leads to myopia and backward displacement of the lens leads to hypermetropia

3. Describe the causes, clinical features, and treatment of irregular astigmatism. (1+2+2 marks)

Irregular Astigmatism

- A type of astigmatism in which the refractive power changes irregularly from one meridian to the other
- It is usually curvatural

Causes

Scarred cornea, subluxated lens due to varying laxity of zonules at different parts of the lens.

Clinical Features

Symptoms

Patients present with distortion of objects, blurred vision, and sometimes polyopia

Signs

- Corneal surface may appear irregular and scarred
- If associated with subluxated lens, there is iridodonesis, phacodonesis, and uneven anterior chamber
- Distortion of mires on placido disc and photokeratoscopy

Treatment

- Rigid contact lenses
- Photorefractive keratectomy
- Penetrating keratoplasty as a last resort

4. Define aniseikonia. State its types. (1+2 marks)

Aniseikonia

Definition

A condition in which the images projected onto the visual cortex by two retinae are of different sizes and shapes.

Types

1. Optical aniseikonia due to anisometropia
2. Retinal aniseikonia may occur due to retinal stretching due to edema
3. Cortical aniseikonia

5. Name the types of anisometropia. (3 marks)

1. **Simple**
   - When one eye is normal and the other is either myopic/hypermetropic/astigmatic
2. **Compound**
   - When both are myopic/hypermetropic/astigmatic of unequal degrees
3. **Mixed**
   - When one is myopic and other is hypermetropic

6. Explain the terms (1 mark each)

A. Far point
B. Near point
C. Amplitude of accommodation

A. Far Point

- Punctum remotum: or far point is the farthest point that is visible
- It is taken as infinity for emmetropic eye, is a real point in front of the retina in myopic eyes, and a virtual point behind the retina in a hypermetropic eye

B. Near Point

- Punctum proximum or near point is the nearest point which is visible to the naked eye
- Its value changes with age

10 years	7 cm
40 years	25 cm
45 years	33 cm

C. Amplitude of accommodation

- The difference between dioptric power needed to focus near point and the far point is called the amplitude of accommodation
- A = P–R

7. Explain how the near point is measured? (3 marks)

- The near point is the nearest point that is visible to the naked eye
- It is measured using the RAF rule (Royal Airforce) (Fig. 1.2.10)
- During the test, the patient must wear his full optical correction
- The instrument consists of a specially designed rule on which centimeters and inches are marked on two sides
- The third side is marked with diopters for measuring NPA, and the fourth side is marked by the appropriate age
- A wing-like structure is present on one end that fits over the nose and rests over the maxillary region
- A mobile rider is present that moves along the length of the rule
- The wing-like end of the instrument is placed on the nose and the rider is moved closer to the eyes till the closest point till which the 6/9 letters on it are clear
- The distance is measured by the markings at which the rider coincides and it is the NPA

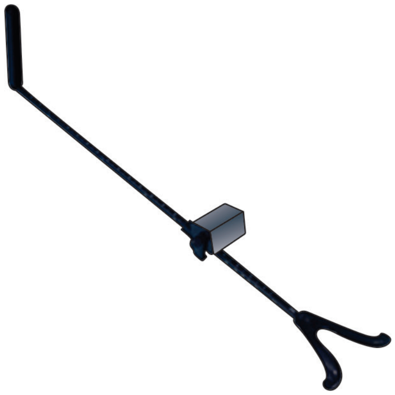


Fig. 1.2.10: RAF rule

8. Explain progressive spectacles. (3 marks)

- Progressive power lenses are also called Varifocal lenses
- They have a progressive increase in the power of the lens from the upper part downwards
- A standard progressive power adds lens is designed to have four zones (Fig. 1.2.11)
  1. Distance vision zone in the upper part
  2. Near vision zone in the lowermost part
  3. Progressive corridor connecting the above two
  4. Blending region on either side.

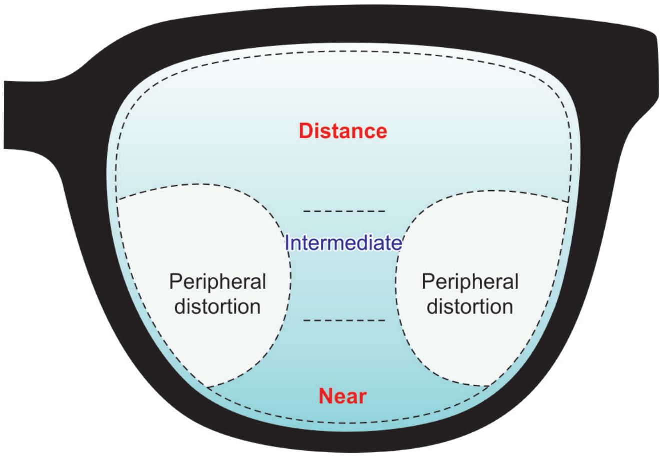


Fig. 1.2.11: Progressive glasses

9. Enumerate the types of contact lenses. (3 marks)

1. Hard contact lens
2. Rigid gas permeable lens
3. Soft lens

10. Enumerate the disadvantages of using aphakic glasses. (3 marks)

- Using highly plus lenses to correct aphakia was the most common method used in the past in developing countries
- It has many disadvantages
  1. Heavy
  2. Magnification up to 30%, can cause diplopia if unilateral
  3. Chromatic and spherical aberration. Spherical aberration causes the pincushion effect
  4. Decreased field of vision
  5. Jack-in-the-box phenomenon or roving ring scotoma
  6. Cosmetically not acceptable by many

11. Enumerate the indications for low vision aids. (3 marks)

Low Vision Aids

A. Macular Disorders: Magnifiers and Telescopes are Useful

1. Albinism
2. Age-related macular degeneration
3. Heredomacular dystrophies
4. Diabetic maculopathy
5. Chorioretinitis
6. Myopic macular degeneration

B. Retinal Dystrophies: Magnifiers with Increased Illumination are Useful

1. Retinitis pigmentosa
2. Choroideremia

12. A patient has been prescribed the following prescription for spectacles.

Right Eye	Left Eye
–3.0D sphere –0.75D cylinder @ 90 degrees	–2.25D sphere

A. State the types of refractive errors in both eyes. (1+1 marks)

B. Define hypermetropia. (1 mark)

A. Types of Refractive Errors in Both Eyes

1. **Right eye:** Compound myopic astigmatism (against the rule)
2. **Left eye:** Myopia

B. Definition of Hypermetropia

A type of refractive error where the light rays traveling from infinity are focused onto a point posterior to the retina. As a result, the image is formed behind the retina.

13. A 14-year-old boy playing cricket gets an injury to his right eye. His lens is subluxated. State the type of refractive error induced and explain its mechanism. (1+2 marks)

Type of Refractive Error

Subluxation of the lens causes myopic astigmatism and is usually irregular.

Mechanism

- Normally, the lens is held tightly by the zonules and the anterior surface (that determines the refractive error) is even, and thus, there is no or minimal astigmatism
- In subluxation (partial loss of zonules), a part of the zonular ring is disrupted

14. Explain the optical phenomenon indicated by the image. (3 marks)

- The image shows a Jack-in-the-box
- Jack-in-the-box phenomenon or roving ring scotoma: a ring-shaped relative scotoma in the midfield between the central field and peripheral field (50–65°) (Fig. 1.2.12)
- Objects in this area are not visible but appear suddenly out of nowhere when the patient moves his eye towards the object or it enters his central or peripheral field
- It happens due to the prismatic effect of the convex lenses

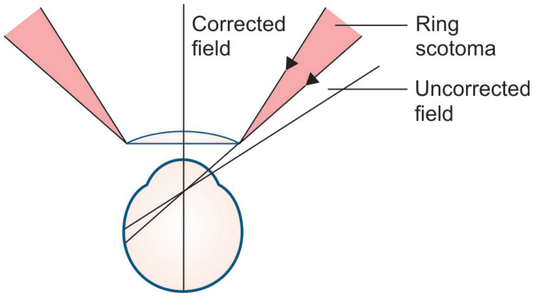


Fig. 1.2.12: Jack-in-the box phenomenon

COMPETENCY

OP1.3 DEMONSTRATE THE STEPS IN PERFORMING THE VISUAL ACUITY ASSESSMENT FOR DISTANCE VISION, NEAR VISION, COLOR VISION, THE PINHOLE TEST, AND THE MENACE AND BLINK REFLEXES

SHORT ESSAYS

1. Explain the steps in visual acuity assessment. (5 marks)

Visual acuity assessment includes the following steps:

1. **Objective refraction**
    - In this method, the amount of refractive error is assessed by either manual or automated methods
    - The manual method is done by a hand-held retinoscopy
    - Retinoscopy is a procedure for objective refraction where a beam of light emanating from a source is shone into the pupil of an individual and moved from one side to the other in both meridia
    - Based on the movement of the reflex within the pupil, certain inferences can be drawn
    - These inferences are based on the presumption that the examiner is seated at 1 m far from the patient

  - Once the type of refractive error is determined, the degree of the refractive error should be calculated
  - It is done by placing progressively higher ‘minus’ lenses in front of the eye when the reflex is moving against the movement till the movement is neutralized or reversed
  - In cases of ‘with-the-movement’ of the reflex, ‘plus’ lenses are placed in ascending progression, till there is the reversal of movement
  - Automated refraction is done by using an autorefractometer

2. **Subjective refraction**
   - This is done by the trial and error method
   - A trial frame is used to place lenses and the distance and near vision based on the readings obtained by objective refraction
   - The patient is asked to read out the Snellen’s chart placed at a distance of 6 m
   - Snellen’s chart has letters or numbers with decreasing size.
   - The biggest of the character is denoted as 6/60; meaning what the patient has to view at 60 m, can be viewed by him at 6m (indicating subnormal vision)
   - Thus, there are various grades like 6/36, 6/24, 6/18/6/12, 6/9, and 6/6
   - This is followed by a near vision assessment with a near vision chart (Jaeger)
3. **Refinement of refraction**
   - Refinement of a sphere is done by Fogging method and Duochrome test
   - Refinement of the cylinder is done by Jackson’s cross-cylinder and Astigmatic fan test
4. **Binocular balancing**
   - This refers to finding any oculomotor imbalances like manifest deviation, convergence insufficiency, etc

2. Describe the principles and procedure of retinoscopy. (2+3 marks)

Retinoscopy

Principles

- In the determination of refraction, the first objective refraction is done to determine the type of refractive error—myopia, astigmatism or astigmatism or a combination and then their magnitude
- This is performed manually by retinoscopy or skiascopy. It is based on the principle that when light is shone into the eye, the direction of movement of the reflex depends on the refractive status of the eye

Results of Retinoscopy (Fig. 1.3.1)

If the shadow moves in the opposite direction, then the refractive power is >1D myopia

In such cases, minus lenses are placed in front of the eye till the movement changes in the same direction

If the shadow moves in the same direction, then it is either emmetropia or hypermetropia or myopia of less than 1D

In such cases plus lenses are placed in front of the eye till there is a change of movement to the opposite direction

If the shadow does not move, then it is the myopia of 1D

Procedure

- The patient is seated in a dark room and asked to focus on Snellen’s chart
- The examiner sits 1m in front of the patient and shines light into the patient’s eye in one direction and noted the movement of the shadow

- Once the type of refractive error is determined, the degree of the refractive error should be calculated
- It is done by placing progressively higher 'minus' lenses in front of the eye when the reflex is moving against the movement till the movement is neutralized or reversed
- In cases of 'with-the-movement' of the reflex, 'plus' lenses are placed in ascending progression, till there is a reversal of movement
- If there is a requirement of use of cycloplegic, a cycloplegic is used

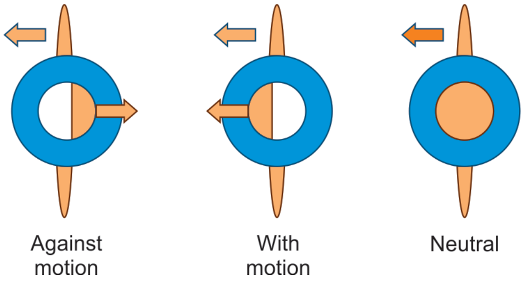


Fig. 1.3.1: Results of retinoscopy

SHORT ANSWERS

1. Demonstrate the ability to identify refractive errors based on retinoscopy findings. (3 marks)

- Retinoscopy is a procedure for objective refraction where a beam of light emanating from a source is shone into the pupil of an individual and moved from one side to the other in both meridia
- Based on the movement of the reflex within the pupil, certain inferences can be drawn.
- These inferences are based on the presumption that the examiner is seated at a distance of 1m from the patient
- If the shadow moves in the same direction, then it is either emmetropia or hypermetropia or myopia of less than 1 D (Fig. 1.3.2)
- If the shadow moves in the opposite direction, then the refractive power is >1D myopia (Fig. 1.3.3)
- If the shadow does not move, then it is a myopia of 1D (Fig. 1.3.4)
- Once the type of refractive error is determined, the degree of the refractive error should be calculated
- It is done by placing progressively higher 'minus' lenses in front of the eye when the reflex is moving against the movement till the movement is neutralized or reversed
- In cases of 'with-the-movement' of the reflex, 'plus' lenses are placed in ascending progression, till there is a reversal of movement

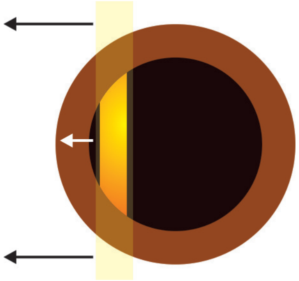


Fig. 1.3.2: With movement

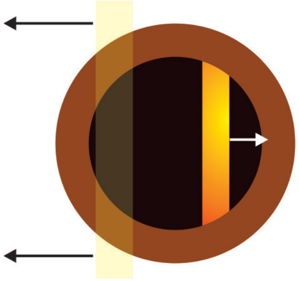


Fig. 1.3.3: Against movement

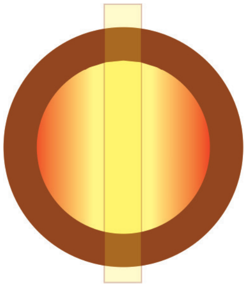


Fig. 1.3.4: Neutralization of reflex

2. Describe refinement of the cylinder after the trial-and-error method. (3 marks)

Refinement of the cylinder is performed using an astigmatic fan and Jackson's cross cylinder

1. **Astigmatic fan (Fig. 1.3.5)**
   - The astigmatic fan consists of a set of radiating lines spaced at 10° apart, with a central panel that contains a 'V' and a set of perpendicular lines.
   - After obtaining the best-corrected visual acuity, a positive sphere is added to the trial frame to induce simple myopic astigmatism
   - Then, the patient is instructed to point out the group of lines that appear clearest and darkest. This gives an idea about the probable axis

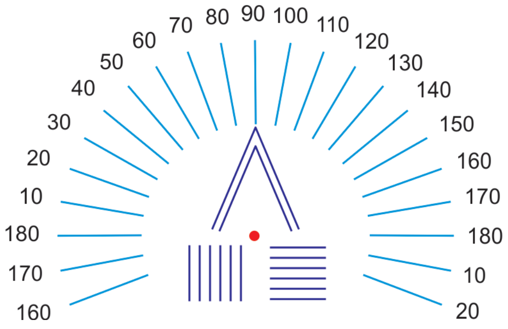


Fig. 1.3.5: Astigmatic fan

2. **Jackson's cross-cylinder**
   - It is a combination of two cylinders of equal strength but in perpendicular meridia (Fig. 1.3.6)
   - The commonly used powers are ±0.25 and ±0.5
   - The patient's best-corrected visual acuity with the sphere is found and placed in the trial frame
   - The cross-cylinder is placed with the 'plus' mark at 90° and followed by 180°
   - If there is an improvement of visual acuity with any of the positions, there is a requirement of a cylinder in that particular axis
   - To refine the axis, the cross-cylinder is placed in a way such that both axes are 45° to the assumed axis.
   - This is followed by flipping the cross-cylinder
   - If the patient notices improvement in either position, the plus cylinder is rotated towards the plus sign of the cross-cylinder and the minus is rotated towards the minus

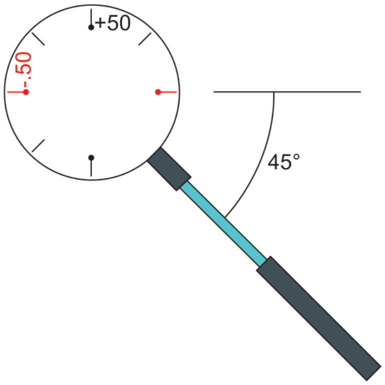


Fig. 1.3.6: Jackson's cross-cylinder

3. Explain the role of a pinhole in refraction? (3 marks)

- The pinhole is a very useful instrument in refraction
- It is nothing but an occluder with a central point-sized opening

Principle

- It is based on the principle that smaller pupils give a good depth of focus and clarity due to smaller aperture
- So, when light rays are made to pass through the pinhole, the central rays only pass through. These rays are not affected by refractive errors which arise due to the curvature of the refractive surfaces (Fig. 1.3.7)

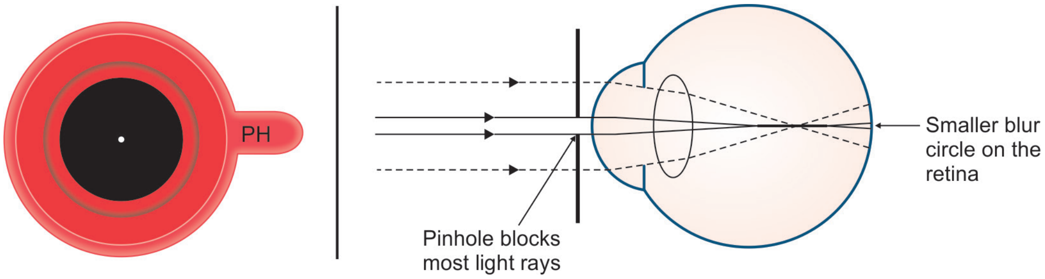

PH
Smaller blur circle on the retina
Pinhole blocks most light rays

Fig. 1.3.7: Principle of pinhole

Use in Refraction

- When the uncorrected visual acuity is less than 6/6, a pinhole is placed in the trial frame. If the visual acuity improves to 6/9 or 6/6/, then we know the cause for blurred vision is a refractive error
- If the visual acuity does not improve with the use of a pinhole, it is more probable that the cause for diminution of vision is some other pathology

4. Explain how menace reflex is elicited? State its clinical implication. (1+2 marks)

Menace Reflex Elicitation

- To perform this test, the patient is seated comfortably and instructed to occlude one eye
- Menace reflex is elicited by suddenly bringing an object/examiner's hand close to the eye of the patient
- The patient with moderately fair vision blinks as a protective response (both the eyelids close)

Clinical Implication

- Menace reflex is a frequently used bedside test for testing visual acuity in patients who cannot take part in conventional visual acuity testing
- It is a type of blink reflex that leads to reflex blinking in response to a fast-approaching object towards the eye
- It is absent in damage to the visual cortex
- It is useful in the rough assessment of vision in children, mentally challenged, and malingerers

5. Explain how blink reflex is elicited? State its clinical implication. (1+2 marks)

Blink Reflex Elicitation

- To elicit it, the examiner stands beside the patient and the patient is advised to look straight ahead
- With a wisp of cotton, the examiner touches the corneal limbus without bringing the cotton wisp into the patient's field of vision (otherwise menace reflex gets elicited)
- This causes bilateral blinking
- The same procedure is repeated on the other side. It can be quantified by using an aesthesiometer

Clinical Implication

- It is an involuntary reflex characterized by blinking of the eyelid followed by corneal stimulation
- It is a bilateral response; the afferent pathway is through the ophthalmic division of the trigeminal nerve and the efferent is through the zygomatic branch of the facial nerve
- The time taken is 0.1 seconds
- It is a defensive mechanism to protect the eye from injury. It forms the basis for testing corneal sensations
- Absent blink reflex is seen in several cases where trigeminal nerve endings are damaged
- Asymmetric blink reflex is seen in facial palsy

6. A 4-year-old was found to have a refractive error of +3D. But he cannot be prescribed glasses right away. Justify the use of cycloplegics in refraction. (3 marks)

- A 4-year-old child with hypermetropia of 3D has been examined in this case.
- Glasses should not be prescribed right away as children have a ciliary tone that masks the actual refractive error
- Cycloplegics are drugs that cause paralysis of accommodation leading to uncovering the actual refractive error that has been corrected by ciliary tone
- They are especially indicated in children, hypermetropes and severe astigmatism to confirm the axis.
- Retinoscopy performed after cycloplegics is called wet retinoscopy

Cycloplegic	Age for use	Dosage	Time of retinoscopy	PMT performed
Atropine (1%)	<5 years	Given as ointment BID for 3 days	3 days	After 3 weeks
Homatropine (2%)	5–8 years	1 drop every 10 minutes for 60 minutes	90 minutes	3 days
Cyclopentolate (1%)	8–13 years	1 drop every 15 minutes, 3 applications	90 minutes	3 days
Topricamide (1%)	Only as a mydriatic	1 drop every 15 minutes, 3 applications	40 minutes	Next day

- After doing a retinoscopy in a cycloplegic state, a post-mydriatic test subjective refraction is performed after the prescribed period
- Correction is only prescribed instead of performed after this test.

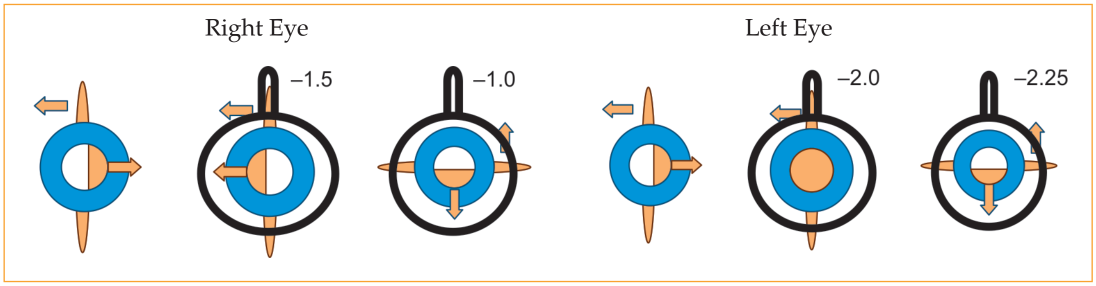
7. Describe the method of using Ishihara's chart. (3 marks)

Method of Using Ishihara's Chart

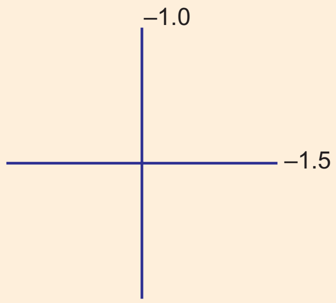
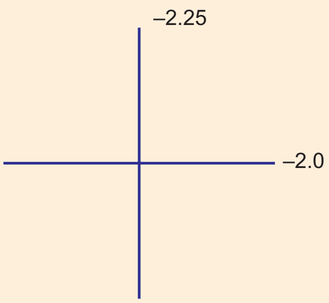
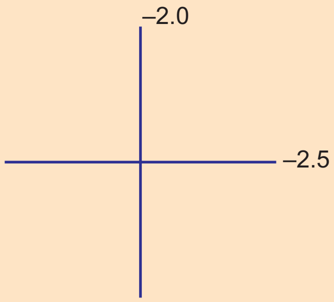
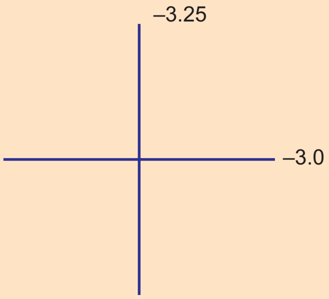
- It was designed by a Japanese scientist and is designed to identify congenital color blindness
- Individuals with color blindness are asymptomatic and see all colors in various shades of grey
- In the Ishihara chart, there are various colored circles that are of various intensities of the same hue within which a number is hidden. Also, the number is made of colored circles of different shades of the same hue

- This is called pseudo-isochromatism—a false impression of being of the same color
- To perform this test, the individual is asked to cover one eye and identify the numbers in the chart. The examiner notes the readings by the individual. This is followed by an assessment of the other eye
- A table is provided at the end of the chart that helps to categorize the type of color blindness

8. A 29-year old man presents with blurred vision for distance. On performing dry retinoscopy at 1m, the following results are seen. Draw a power cross for the same and deduce the power of his glasses. (2+3 marks)



Retinoscopy values performed at the vertical and horizontal meridian are shown in the images

Feature	Right Eye	Left Eye
Power cross		
After deduction of –1 for distance		

Prescription	–2.0D sphere –0.5D cylinder at 90°	–3.0D sphere –0.25D cylinder at 180°
Diagnosis	Compound myopic astigmatism (against the rule)	Compound myopic astigmatism (with the rule)

9. A 40-year old carpenter wants presbyopic correction. He is emmetropic for distance. His NPA (near point of accommodation) is found to be 50 cm and his working distance is 33 cm. Calculate the amount of 'plus' to be prescribed. Explain the impact of age on accommodation. (2+1 marks)

Amount of Plus

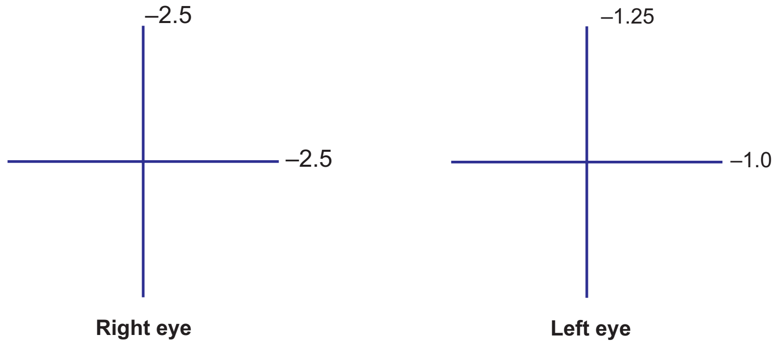
- For prescribing presbyopic correction, the patient's occupational requirement, ambient lighting, and NPA need to be considered
- NPA, in this case, is 50 cm hence the accommodating power necessary to focus adequately is 100/50 (reciprocal of focal length in meter) = 2D
- His working distance is 33 cm (0.3 m). Hence the accommodating power necessary to focus on the object is 100/33 = 3D
- So, the necessary add for presbyopic correction is 3 – 2 = 1D. However, while prescribing presbyopic correction, one-third of the necessary accommodating power (3D) is decreased to provide comfortable viewing. One-third of 3D is 1D
- Thus, this patient does not need presbyopic correction

Effect of Age on NPA

The NPA recedes with age due to a decrease in the elasticity of the lens capsule

Age (years)	Distance (cm)
10	7
20	10
30	14
40	20
50	40

10. A 23-year-old person came with a history of difficulty in seeing distant objects. On retinoscopy (at 1m), the following measurements were noted in both eyes. Determine the type of refractive error and its value. (1+2 marks)



Type of Refractive Error

Both eyes have a retinoscopy value of more than –1. So, there is myopia in both eyes.

Value of refractive Error

- *Value of refractive error in the right eye:* –2.5 – 1.0 in both horizontal and vertical meridian = –3.5D
- *Value of refractive error in the left eye:* –1.25 – 1.0 in both horizontal and vertical meridian = –2.25D

COMPETENCY

OP1.4 ENUMERATE THE INDICATIONS AND DESCRIBE THE PRINCIPLES OF REFRACTIVE SURGERY

LONG ESSAY

1. A 20-year-old patient with –9DS in the right eye and –8.5DS in the left eye wants to get rid of her spectacles.

A. Name two refractive surgeries that can be useful in this patient. (2 marks)
B. Classify various corneal and lenticular refractive procedures. (5 marks)
C. Explain the role of femtosecond LASER in refractive surgeries. (3 marks)

A. Refractive Surgeries that can be Useful

1. Photorefractive keratectomy
2. Laser-assisted *in situ* keratomileusis (LASIK)

B. Classification of Refractive Procedures

Corneal procedures/keratorefractive procedures	Lens-based procedures
Incisional • Radial keratotomy • Astigmatic keratotomy • Arcuate keratotomy • Limbal relaxing incisions • Hexagonal keratotomy	Phakic • Anterior chamber • Iris fixated or Worst Iris claw lens • Posterior chamber phakic IOLs
Excimer LASER • Surface ablation ▪ Photorefractive keratectomy ▪ Laser subepithelail keratomileusis ▪ Epipolis LASIK • Lamellar ▪ Laser *in situ* keratomileusis ▪ Femtosecond LASIK ▪ SMILE	Pseudophakic • Refractive lens exchange with multifocal/ toric IOL or accommodating IOL
Non-LASER lamellar • Intracorneal ring segments Collagen shrinkage • Conductive keratoplasty • LASER thermokeratoplasty	Combined (Corneal and lenticular) • Bioptics

C. Role of Femtosecond LASER in Refractive Surgeries

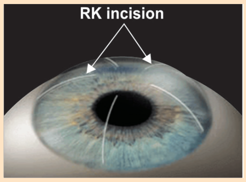
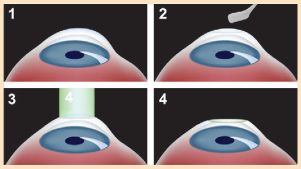
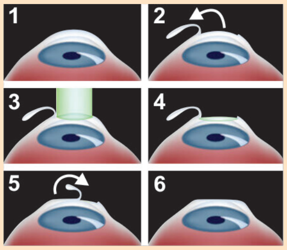
- Femtosecond LASER is an infrared laser and acts by photo disruption
- It is also called 'ultrafast LASER' as the wavelength is 10^{-15} m

- As a result, the energy delivered is much lesser with higher precision. There is lesser damage to the surrounding tissues
- Their uses in refractive surgeries are:
  1. In conventional LASIK, the corneal flap is created using a microkeratome. In femtosecond LASER-assisted LASIK, the flap is raised using LASER. Thus, it is a more delicate process
  2. Channel creation for intrastromal ring segments
  3. Astigmatic keratotomy
  4. Small incision lenticule extraction
  5. Intrastromal presbyopia correction

SHORT ESSAYS

1. Compare and contrast radial keratotomy, photorefractive keratectomy, and LASIK for correction of myopia. (5 marks)

Comparison of Radial Keratotomy, Photorefractive Keratectomy, and LASIK

Feature	Radial keratotomy (Fig. 1.4.1)	Photorefractive keratectomy (Fig. 1.4.2)	LASIK (Fig. 1.4.3)
Type of procedure	It is an incisional technique	Surface laser ablation	Lamellar laser ablation
Principle	Radial incisions are made up to 90% thickness of the cornea leaving a central 4 mm of the clear cornea. When these incisions heal by scarring, they flatten the cornea	The cornea is de-epithelialized and LASER ablation is done in the central part of the cornea that causes corneal flattening and thus correction of myopia	A superficial flap of the cornea (epithelium, basement membrane + superficial stroma) is raised with the help of a microkeratome or femtosecond LASER. The corneal bed is ablated and the flap is reposited
	 **Fig. 1.4.1:** Radial keratotomy	 **Fig. 1.4.2:** Photorefractive keratectomy	 **Fig. 1.4.3:** LASIK
Present-day use	It was a breakthrough in the early 80s, in which many myopic eyes were corrected with this procedure. It is obsolete now	Useful in thin corneas where LASIK is contraindicated Useful in the scarred cornea, epithelial basement membrane dystrophies, and people involved in contact sports	Most commonly used in the present day Novel improvements like epi-LASIK, wavefront-guided LASIK are gaining more importance
Range of myopia	Low range of myopia	Up to –14D	Up to –14D

Contd.

Feature	Radial keratotomy (Fig. 1.4.1)	Photorefractive keratectomy (Fig. 1.4.2)	LASIK (Fig. 1.4.3)
Disadvantages	• Diurnal variation of power due to increase in thickness in waking hours • Hypermetropic shift • Glare • Cornea is weakened • Can correct only low grades of myopia	• Central corneal haze and delayed healing	• Overcorrection can occur if the wound healing is delayed • Under-correction if wounds heal quickly • Decentered ablation • Flap dislocation • Dry eye due to injury to corneal nerves • Infectious keratitis • Keratectasia

2. Explain the indications, principles, and the procedure of LASIK.

(1+1+3 marks)

LASIK

Indications

1. Myopia up to –14D
2. Hypermetropia up to +6D
3. Astigmatism up to 5D
4. Presbyopia: Monovision LASIK, presbyopia bifocal LASIK

Principle

- A superficial flap of the cornea (epithelium, basement membrane + superficial stroma) is raised with the help of a microkeratome or Femtosecond LASER
- The corneal bed is ablated and the flap is reposited

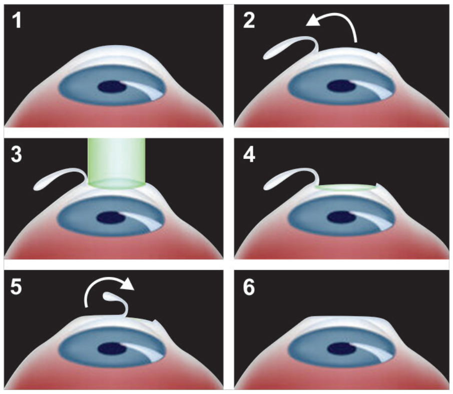


Fig. 1.4.4: LASIK procedure

Procedure (Fig 1.4.4)

- After a thorough examination to rule out contraindications for LASIK, and stable refraction for a year, the patient is taken up for LASIK procedure
- The amount of tissue to be ablated is calculated before the procedure based on the Munnerlyn formula

$$\text{Ablation depth} = \frac{\text{Diopter of myopia} \times (\text{Optical diameter})^2}{3}$$

- Topical anesthesia is applied, and the patient is instructed to view at a fixation light and to remain still
- A suction ring is applied, and the microkeratome creates a flap of the superficial cornea. The motor of the microkeratome oscillates the blade at the rate of 6000 cycles per minute
- Instead of microkeratome, femtosecond LASER can be used to create the flap

- The stromal bed is ablated with excimer LASER and then the flap is reposited and a bandage contact lens is placed
- The patient is started on topical antibiotics and steroids

3. Describe the refractive surgeries available for hypermetropia. (5 marks)

1. **Hyperopic LASIK**
    - The procedure of LASIK is similar to that performed for myopia: After calculation of the amount of tissue to be ablated, the flap is created using a microkeratome or femtosecond LASER
    - The mid-peripheral cornea is ablated using Excimer LASER to create steepening of the central cornea thus causing a myopic shift in refraction (Fig. 1.4.5)
2. **Hyperopic PRK**
    - The cornea is de-epithelialized and LASER ablation is done in the mid-peripheral part of the cornea that causes central corneal steepening and thus correction of hyperopia
3. **Refractive lens exchange**
    - This method is reserved for very high degrees of hypermetropia
    - It is a cataract surgery before the development of the cataract. The clear lens is extracted through small incisions created at the cornea or limbus and an appropriate IOL is placed in the posterior capsular bag

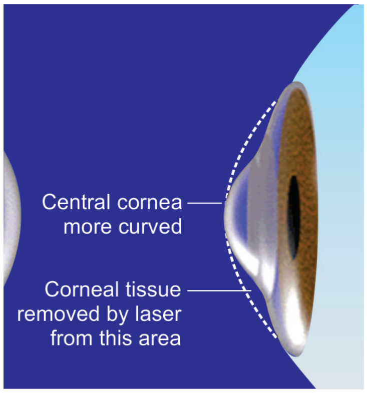

Central cornea more curved
Corneal tissue removed by laser from this area

Fig. 1.4.5: Hyperopic LASIK

4. **Phakic intraocular lens**
    - In this procedure, an intraocular lens is placed in the posterior chamber (PC-PIOL), anterior chamber (AC-PIOL), or iris-claw lenses are used
    - This procedure demands a minimum depth of anterior chamber of 3 mm at the center and a healthy endothelium
5. **Holmium LASER thermoplasty**
    - The Ho:Yag LASER produces light in the infrared region and is used to cause heat production
    - When applied to the cornea, the heat generated by the tissues causes shrinkage and thus alters the shape of the cornea
    - These burns are applied in the mid-periphery and thus cause central steepening. This method has become obsolete now
6. **Conductive keratoplasty**
    - Galvanic current is used to flatten the mid-peripheral cornea
    - As a result, the central cornea is steepened

4. A 50-year-old actress has presbyopia and does not want to wear glasses. She requests refractive surgery for the same. Describe the refractive surgeries that you can offer her. (5 marks)

Treatment of presbyopia can be given as monovision or binocular procedures

A. Monovision

There is no binocularity following these techniques

1. **Monovision conductive keratoplasty**
   - In this technique, galvanic current is applied in the mid-periphery of the cornea to cause central corneal steepening
   - Thus one eye becomes slightly more myopic and presbyopia is corrected
2. **Monovision LASIK**
   - One eye is corrected for distance vision and the other is corrected for near vision
3. **Monovision with IOL**
   - Clear lens extraction with myopic correction in one eye and hyperopic correction in the other is the principle of this procedure

B. Binocular Surgeries

1. **Bifocal LASIK or LASIK PARM**
   - This is also called the Avalos technique
   - Here the cornea is ablated in such a way that there are two concentric zones: One for distance vision and the other for near vision
   - The central cornea is flatter, and the mid-peripheral cornea is steeper
   - Central presbyLASIK is a method in which the central cornea is steepened, and the mid-peripheral cornea is ablated for distance (Fig. 1.4.6)

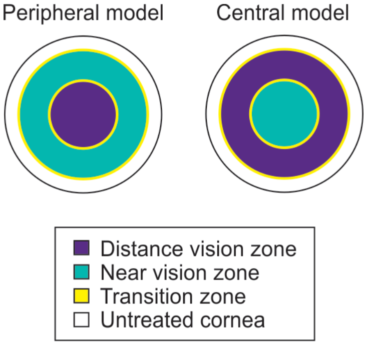


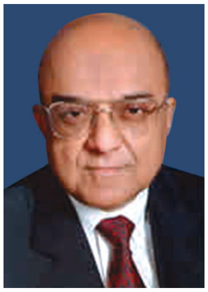
Fig. 1.4.6: Bifocal LASIK

2. **Multifocal LASIK**
   - Based on a similar principle as multifocal IOLs, the cornea is divided into several concentric zones of varying power. The central zone is meant for near vision
3. **Multifocal or accommodating IOL**
   - Clear lens extraction is followed by implantation of a multifocal or accommodating IOL
4. **Corneal inlays**
   - A pocket is created in the superficial cornea using Femtosecond LASER and intra-corneal rings are placed
   - These rings help to converge the divergent rays and thus help in presbyopia

SHORT ANSWERS

1. This Indian Scientist based in the USA discovered excimer LASER. Identify him.

Describe the principle of excimer laser in refractive surgeries. (1+2 marks)



Given Scientist

Rangaswamy Srinivasan discovered the use of Excimer laser in re-shaping the cornea.

Principle of Excimer Laser

- The word ‘excimer’ is derived from the combination of two words: excited dimer. The dimer is made of a noble gas such as argon or helium combined with a halogen (ArF, HeBr)
- It has a wavelength of 193 µm and lies in a high ultraviolet ray range

- It cannot pass through the cornea and thus is safe to the eye. Also, it is non-mutagenic
- This LASER acts by breaking the chemical bonds between the tissue, a process known as photoablation
- They break the carbon-carbon and carbon-nitrogen bonds that form the backbone of the collagen framework
- This property is used to re-mold the shape of the cornea to achieve the desired refractive outcome
- Properties of excimer that make it suitable for refractive surgeries:
    1. High precision, low collateral damage
    2. Low penetrability
    3. Non-mutagenicity

2. Explain the procedure of SMILE or small incision lenticule extraction. (3 marks)

- Small incision lenticule extraction or SMILE is also called as ReLex (refractive lenticule extraction) (Fig. 1.4.7)
- It is a relatively new, Femto-assisted, minimally invasive refractive surgery
- In this technique, after the calculation of lenticule to be removed, the lower interface of the lenticule is created first
- Followed by this, the upper interface or the 'cap' of the lenticule is created

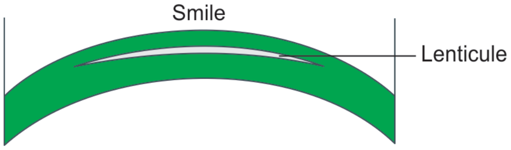


Fig. 1.4.7: Procedure of SMILE

- Then, a superotemporal 2–3 mm long tunnel incision is made to connect the lenticule to the surface of the cornea
- Then the patient is moved to the surgical microscope and the lenticule is extracted with the help of retinal micro-forceps

3. Define corneal topography. Enumerate two indications for the same. (3 marks)

Corneal Topography

Definition

- Study of the surface of the cornea
- It is a computer-assisted tool that gives a three-dimensional color-coded curvatural map of the cornea

Indications

1. Before performing a refractive surgery
2. Diagnosis and follow-up of keratoconus

4. Enumerate six complications related to LASIK. (3 marks)

Complications of LASIK

1. Microkeratome-related complications: The creation of free flap, too thin flap, buttonholing of flap
2. Flap folds or striae
3. Infectious keratitis
4. Traumatic flap dislocation
5. Diffuse lamellar keratitis
6. Post-LASIK ectasia

5. Describe the technique of arcuate keratotomy. (3 marks)

Arcuate Keratotomy

- It is an incisional technique that was used previously for the correction of astigmatism.
- Presently, it finds its use in the correction of post-keratoplasty astigmatism
- It is based on the principle of coupling: When one meridian of the cornea is flattened, the other meridian that is perpendicular to the former becomes steep (Fig. 1.4.8)
- In this technique, after applying topical anesthesia, a diamond blade is used to make 95% depth arcuate incisions in the steep meridian in the mid-peripheral cornea
- The length, depth, and site of the incision determine the amount of correction
- Longer, deeper, and more central the incision, the greater is the astigmatism correction

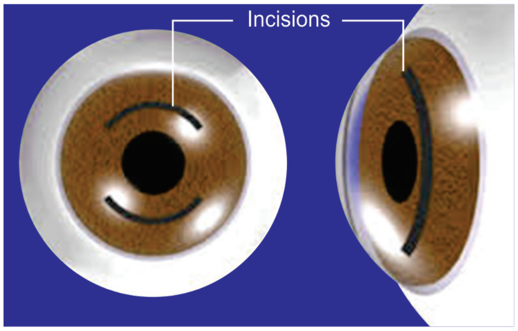


Fig 1.4.8: Arcuate keratotomy

6. What are intrastromal ring segments? List two advantages and disadvantages over LASIK. (2+1 marks)

Intracorneal Ring Segments

- These are PMMA rings that are placed in the mid-peripheral cornea
- They act by displacing the central lamellar bundles and cause central flattening

Advantages

1. No flap related complications
2. Reversible
3. Useful in thin corneas

Disadvantages

1. Can correct only low grades of myopia
2. Can induce astigmatism

7. Enumerate the mechanisms of LASER in refractive surgery with examples. (3 marks)

Type of tissue interaction	Mechanism	Clinical utility
Photoablation	Breaks chemical bonds using LASER	Excimer LASER used for photorefractive keratectomy LASIK (wavelength 193 µm)
Photodisruption	Tissue is transformed into plasma	Femtosecond LASER (wavelength 1053 µm)
Photothermal effect	LASER is absorbed by tissue releasing heat which causes collagen shrinkage	Holmium:YAG used in thermoplasty

8. Enumerate three refractive surgeries available for astigmatism. (3 marks)

1. **Incisional techniques**
    - Arcuate keratotomy
    - Limbal relaxing incision
2. **LASER assisted**
    - Astigmatic LASIK
3. **Intraocular surgery**
    - Lens extraction with toric IOL

COMPETENCY

OP1.5 DEFINE, ENUMERATE THE TYPES AND THE MECHANISM BY WHICH STRABISMUS LEADS TO AMBLYOPIA

SHORT ESSAYS

1. Define amblyopia. Explain its types. (1+4 marks)

Amblopia

Definition

- A reduction in the best-corrected visual acuity of one or both eyes that cannot be attributed to a particular structural abnormality of the eye
- Clinically it is defined as visual acuity of 6/12 in both eyes or a difference of 2 Snellen lines between the two eyes.

Types

1. **Stimulation deprivation amblyopia**
    - Was called "amblyopia ex anopsia"
    - Due to pathology of ocular media in formative years
    - Congenital cataract
    - Corneal opacity
    - Severe ptosis
2. **Anisometropic amblyopia**
    - Due to a difference in refractive error, which remains uncorrected
    - Myopia: >4D (less common)
    - Hypermetropia: 1–2D (more common)
3. **Ametropic amblyopia**
    - Bilateral uncorrected refractive error
    - Bilateral hypermetropia >5D and myopia >8D causes amblyopia
4. **Strabismic amblyopia**
    - Uniocular amblyopia in the squinting eye
5. **Meridional amblyopia**
    - High degrees of astigmatism in one or both eyes remain uncorrected.
    - Unilateral astigmatism >1.5D and bilateral astigmatism >2.5D causes meridional amblyopia.

2. Explain stimulation deprivation amblyopia. Give 4 causes for the same. (1+4 marks)

Stimulation Deprivation Amblyopia

- Condition in which one or both eyes have a reduction in visual acuity due to a pathology that blocks the entry of light into the eye and thus prevents the 'stimulation' of the retina for normal development of the visual system in the amblyogenic period, i.e., 6 weeks after birth to 5 years (Fig. 1.5.1)
- It is more severe when unilateral and occurs at the age of 6 weeks to 18 months. This period is termed the critical period of visual development
- Thus, even if the cause for visual deprivation is corrected after the amblyogenic period, the vision is not regained
- Stimulation deprivation causes significant anatomical changes at the cortical level and is the most severe of all forms

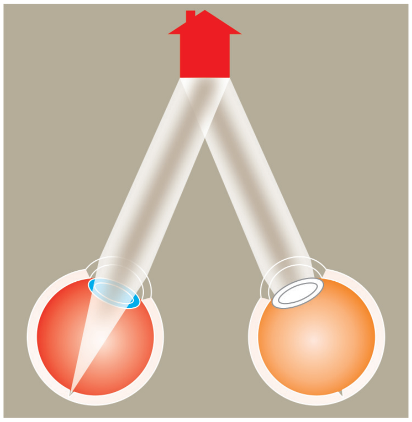


Fig. 1.5.1: Mechanism of stimulation deprivation amblyopia

Causes

1. Corneal opacities and cloudiness are congenital or acquired at a very young age
2. Congenital cataract
3. Ocular trauma leading to corneal or lenticular opacity
4. Congenital ptosis that covers the pupil
5. Nystagmus disorders that prevent foveation
6. Congenital retinal disorders

3. A 3-month-old child is diagnosed to have a unilateral central congenital cataract. Her parents want to wait until she turns one year of age as they feel the child is too young for surgery.

A. Explain the consequences of waiting for more time concerning visual acuity? (2 marks)

B. Enumerate the clinical features of this condition. (3 marks)

A. Consequences of Waiting for More Time Concerning Visual Acuity

- In this case, the child has a unilateral cataract, which is central
- As a result, the number of light rays entering the eye is decreased
- There is a high chance of developing stimulation deprivation amblyopia
- As the parents want to wait for a longer time, it is advisable to counsel parents in such cases regarding the development of amblyopia, especially as the cataract is unilateral
- Even if the child undergoes cataract extraction at a later date, a certain amount of amblyopia might have already set in and thus the postoperative best-corrected visual acuity is expected to be lower than usual
- This will affect the binocularity of the child

B. Clinical Features of Amblyopia

- Visual acuity is reduced
- It does not improve even after the offending factor is removed/corrected. In this case, congenital cataract
- Recognition acuity is more affected than resolution acuity

- Visual acuity when tested through a neutral density filter improves by one or two lines in amblyopia, in contrast to organic lesions
- Crowding phenomenon: Visual acuity is better when single letters are presented
- The fixation pattern may be central or eccentric
  - The degree of amblyopia in eccentric fixation is proportionate to the distance of the eccentric point from the fovea
  - In simple terms, amblyopia tends to more severe in a larger degree of squint

4. Explain the principles of treatment of amblyopia. (5 marks)

- Most of the treatment patterns have been adopted following the PEDIG (Pediatric Eye Diseases Investigator Group) study
- During the critical period, the steps in correction or treatment of amblyopia are:
  1. Removal of the offending agent like congenital cataract, corneal opacity, correction of squint, or correction of refractive error
  2. This is followed by increasing the use of the amblyopic eye by either occluding the healthy eye or causing cycloplegia for a limited number of hours per day. The "lazy eye" is put to use by restricting the use of the other eye by various mechanical and pharmacological methods
     - Patching or occlusion: This can be accomplished by using an adhesive patch or tape across the eye. Using ground glass spectacles is another method
     - Atropinization is the use of atropine in the sound eye so that there is cycloplegia and the child is forced to use the amblyopic eye. Studies have found occlusion to bring better results
  3. The occlusion can be done full-time or part-time (4–6 hours per day) and both are equally effective
     - However, the latter is preferred to prevent occlusion amblyopia in the sound eye
     - During the occlusion period, the child is made to have both outdoor and near vision work
     - The schedule is patching amblyopic eye: Sound eye in the number of days is given below:

<2 years	2:1
3 years	3:1
4 years	4:1
5 years	5:1

  4. Pharmacological manipulation using citicoline, levodopa-carbidopa have been found to generate new visual circuits and have found good results

SHORT ANSWERS

1. Define the following terms. (1 marks each)

A. Crowding phenomenon
B. Meridional amblyopia
C. Eccentric fixation

A. Crowding Phenomenon

- Visual acuity is better when tested with a chart that has single letters as opposed to the regular Snellen's chart
- This is characteristic of amblyopia

B. Meridional Amblyopia

- When there is a high degree of astigmatism that remains uncorrected, usually more than 1.5D in unilateral cases and more than 2.5D in bilateral cases

C. Eccentric Fixation

- When an extrafoveal point (a point on the retina outside the fovea) takes up fixation due to strabismus, it is called eccentric fixation.
- It is a mechanism to avoid confusion and diplopia

2. How does strabismus lead to amblyopia? (3 marks)

- In squint or strabismus, there is a loss of parallelism of eyes
- As a result, the images formed from both eyes are different
- This causes either confusion or diplopia
- More than diplopia, it is the confusion that causes a decreased response from one retina and this inhibition occurs at the level of the cortex when the strabismus is long-standing, and the eye is still in amblyogenic age
- In such a condition, the brain is capable of restructuring visual circuits that in turn lead to amblyopia
- The eye that is squinting will be suppressed as the image that is exactly in line with the visual axis (as in the straight eye) is chosen as the correct image based on feedback mechanisms by the brain
- Hence, this leads to amblyopia

3. Demonstrate the understanding of the term suppression with diagram. (3 marks)

- Suppression is a phenomenon characterized by temporary active cortical inhibition of images from the squinting eye
- It is seen when both eyes are open. When the nonsquinting eye is occluded, the squinting eye takes up fixation and suppression disappears
- It is due to two different objects projected onto the visual cortex from two eyes. Suppression is an effort to avoid confusion
- The mechanism is eccentric fixation (Fig. 1.5.2)
- Clinically suppression can be tested using the Worth 4 dot test, 4PD base out prism test, and a synoptophore

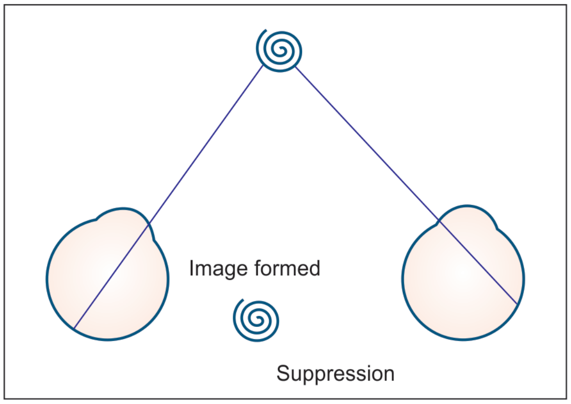


Fig. 1.5.2: Suppression with eccentric fixation

4. Describe occlusion therapy in amblyopia. (3 marks)

- The principle of occlusion therapy is increasing the use of the amblyopic eye by occluding the healthy eye for a limited number of hours per day
- The "lazy eye" is put to use by restricting the use of the other eye by various mechanical methods

- Patching or occlusion: This can be accomplished by using an adhesive patch or tape across the eye. Using ground glass spectacles is another method
- The occlusion can be done full-time or part-time (4–6 hours per day) and both are equally effective. However, the latter is preferred to prevent occlusion amblyopia in the sound eye
- During the occlusion period, the child is made to have both distance and near work
- The schedule is patching amblyopic eye: Sound eye in the number of days is given below:

< 2 years	2:1
3 years	3:1
4 years	4:1
5 years	5:1
6 years	6:1

Prognosis with Occlusion

- Occlusion is the best form of treatment at present when compared to penalization (use of atropine) or pleoptics
- Better results are found in children less than 7 years of age, although improvement has been found until 13 years of age
- Results are better if the visual acuity is good, to begin with, and thus the duration of treatment required is also less
- If there is no clinical improvement in 6 months, there is probably no further improvement expected

5. A 3-year-old child has visual acuity of 6/6 in her right eye, and 6/36 in the left eye. On performing retinoscopy, there is a refractive error of +4D in the left eye. Visual acuity is correctable to 6/18 with respective corrections.

A. State the type of amblyopia in this child. (1 mark)

B. Explain the regimen of occlusion therapy for this child. (2 marks)

A. Type of Amblyopia

This child has anisometropic amblyopia in the left eye.

B. Regimen of Occlusion Therapy

- Since the child is 3 years old, she needs occlusion of the right eye for 2 hours per day for 3 days followed by occlusion of the left eye for 2 hours for one day (3:1)
- During the period of occlusion, the child is made to do activities that engage her in distant and near vision. This is continued for around 6 months, by when improvement is expected

SHORT ESSAYS (MISCELLANEOUS)

1. Describe Listing's reduced eye and its features. Explain its use in ophthalmology. (3+2 marks)

Listing's Reduced Eye

Listing came up with reduced eye with only 4 cardinal points (Fig. 1.M.1).

Anterior focal point	15.7 mm in front of the anterior surface of the cornea
Posterior focal point	24.4 mm behind the anterior surface of the cornea
Principal point	1.5 mm behind the anterior surface of the cornea
Nodal point	7.2 mm behind the anterior surface of the cornea (posterior capsule of the lens)
Anterior focal length	15.7 mm + 1.5 mm = 17.2 mm
Posterior focal point	24.4 mm –1.5 mm = 22.9 mm

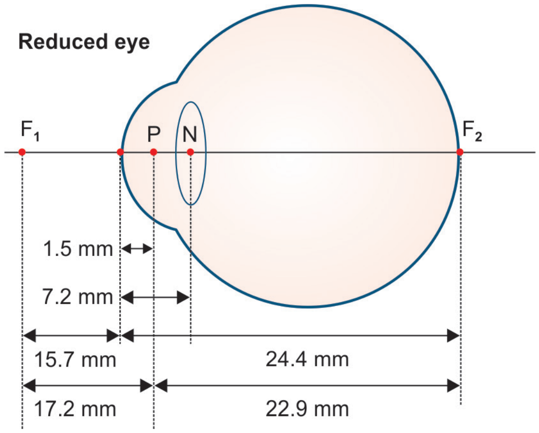


Fig. 1.M.1: Listing's reduced eye

Use in Ophthalmology

1. The concept of the reduced eye helps to understand the optics of various refractive errors like myopia, hypermetropia, and astigmatism
2. By using these points, the total power of the eye could be calculated
   Power of eye = refractive index of air/anterior focal length in meters
   $= 1 \times 1000/17.2 = 58.2\text{D}$
3. Helps to understand the visual angle and the retinal image in various situations

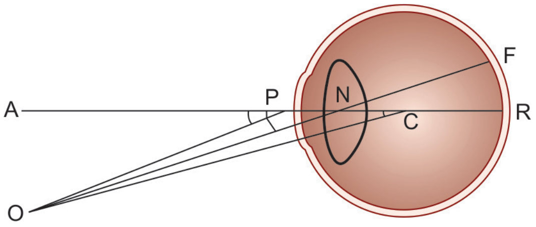


Fig 1.M.2: various angles of the eye

2. Enumerate various angles and axes of the eye as an optical system.

(1.5+1.5 marks)

Angle and Axes of the eye (Fig 1.M.2)

Optical axis	AR	An imaginary line passing through the centre of the cornea, the centre of the lens and then meets the retina nasal to the macula
Visual axis	ONF	An imaginary line passing from the object of interest, nodal point, and the fovea
Fixation axis	OC	An imaginary line joining fixation point and the centre of rotation
Angle alpha	ONA	The angle between the optical axis and visual axis at the nodal point

Contd.

Angle kappa	OCA	The angle between the optical axis (AR) and fixation axis (OC) at the center of rotation of the eyeball
Angle gamma	OPA	The angle between the visual axis (OF) and pupillary line (AP). It is the only measurable parameter Positive angle kappa results in pseudo-exotropia (very high hypermetropia) Negative angle kappa results in pseudo-esotropia (very high myopia)

3. Explain the uses of prisms in ophthalmology. (3 marks)

- Prisms are of great use in ophthalmology
- It is a refracting medium with two surfaces that meet at an angle
- It has an apex and a base (square)
- The angle of the prism is the angle between the two refracting surfaces

Uses in Ophthalmology

1. Measurement of squint
    - Used to detect the amount of squint, to treat small degrees of squint by using a prism bar
    - The prism bar is made of increasing strengths of prisms attached in the form of a bar
    - Light reflex in the cornea is used to assess the amount of squint by placing the prism in front of the deviating eye
2. To detect malingerers by inducing diplopia:
    - A vertical prism of high strength is placed in front of one eye and light is shone
    - If the patient sees two lights, then he is malingering
    - It is a simple and objective test
3. To diagnose and measure fusional reserve in microtropia
    - A 4PD prism is used to detect a very small amount of squint
4. In ophthalmic microscopes, gonioscope, applanation tonometers, and keratometer, prisms are used
5. To eliminate diplopia in reversible and irreparable causes of diplopia, prisms are used. Also, they are used in develop fusional reserve
6. It can be prescribed as a low vision aid for patients with tubular vision or decreased visual field like advanced glaucoma, retinitis pigmentosa, hemianopia where the movements of the eye should be limited

4. Describe various optical aberrations of the eye. (3 marks)

Optical Aberrations of the Eye

1. **Diffraction**
    - When light passes through a small aperture, the light rays produce alternating bands of light and dark bands due to the bending of light rays
    - When the pupil is less than 2 mm, diffraction is highest
2. **Spherical aberrations**
    - It occurs because the spherical lens refracts peripheral rays more strongly than paraxial rays which in the case of a convex lens brings the more peripheral rays to focus closer to the lens (Fig 1.M.3)
    - They present as halos around objects
    - Physiologically, spherical aberrations are kept minimum due to the following factors
        - Pupil size between 3–5 mm. It increases with pupil size

- The Refractive index of the center of the lens is higher than at the periphery
  - o Spherical aberrations increase after surface ablation and LASIK, for myopia and hyperopia. It is dependent on the size of the pupil

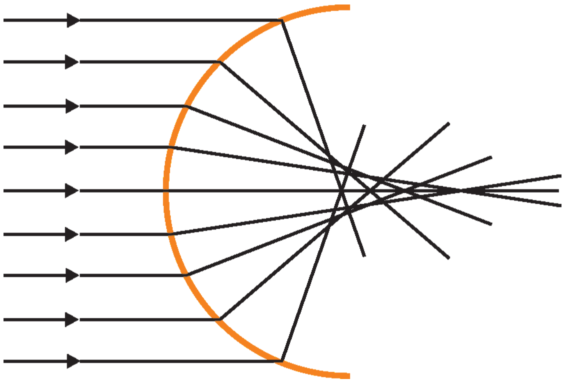


Fig. 1.M.3: mechanism of spherical aberration

3. **Chromatic Aberration**
   - The blue light is focused anteriorly compared to the red light
   - As a result, an emmetropic eye is slightly myopic to blue light and hypermetropic to red light
   - It forms the basis of the duochrome test which is performed at the end of subjective refraction
   - If the patient sees the green light more clearly, either my myopia is over-corrected or my hyperopia is under-corrected
   - If he sees red light more clearly, the inverse is true
   - A myopic should never be over-corrected and a hypermetropic should be fully corrected

5. Describe the etiology, clinical features, and management of anisometropia. (1+2+2 marks)

Anisometropia

- It is a condition in which both eyes have different refractive powers
- It is clinically defined as a difference of more than 2.5D is taken as anisometropia, as up to 2.5D can be tolerated by the cortex
- Up to 4D can be tolerated based on individuals, more than 4D cannot be tolerated

Etiology

- It can be congenital, i.e., if the growth of either eye has different rates, there will be resultant anisometropia
- Acquired anisometropia is following cataract extraction if the eye is left aphakic or an IOL of a wrong power is placed

Clinical Features

- In anisometropia, binocularity is at stake. As a result, stereopsis is compromised
- If one eye has very low vision compared to the other, the fellow eye is used as the fixing eye and the eye with lower vision becomes amblyopic
- If one eye is myopic and the other is hypermetropic, there is alternate vision

Management

- Spectacles for lower degrees of anisometropia, as there will be associated minification/magnification which can lead to aniseikonia
- Contact lenses are advised for higher degrees of anisometropia
- Refractive surgeries for the affected eye/eyes

CHAPTER

2

Lids and Adnexa, Orbit

COMPETENCY

OP2.1: ENUMERATE THE CAUSES, DESCRIBE AND DISCUSS THE AETIOLOGY, CLINICAL PRESENTATIONS AND DIAGNOSTIC FEATURES OF COMMON CONDITIONS OF THE LID AND ADNEXA INCLUDING HORDEOLUM EXTERNUM/ INTERNUM, BLEPHARITIS, PRESEPTAL CELLULITIS, DACRYOCYSTITIS, HEMANGIOMA, DERMOID, PTOSIS, ENTROPION, LID LAG, LAGOPHTHALMOS

LONG ESSAYS

1. A 70-year-old man presents with a foreign body sensation in both eyes for 5 months. On examination, the lid is found to be rolled inwards. A clinical diagnosis of entropion is made.

A. Classify entropion based on etiology. (2 marks)

B. Draw a neat, labeled diagram of the cross section of the lid and enumerate the glands in the lids. (5 marks)

C. State the sequelae of entropion. (1 mark)

D. Explain the grading of entropion. (2 marks)

E. Describe the surgical management of various types of entropion with neat and labeled diagrams. (5 marks)

A. Classification of Entropion

1. **Congenital entropion**
    - Eyelid is inward rotated since birth due to disinserted lower lid retractors
2. **Cicatricial entropion**
    - Due to fibrosis of the posterior lamellae of the lids leading to shortening and inturning of the lid margin
    - It is seen as a sequel to trachoma, membranous conjunctivitis, chemical burns, pemphigus, and Stevens–Johnson syndrome
3. **Spastic entropion**
    - It occurs due to spasm of the orbicularis muscle in patients with chronic irritative corneal conditions or after tight ocular bandaging

4. **Senile (involutional) entropion**
   - Usually affects the lower lid
   - The anatomical factors that contribute to involutional entropion are:
     i. Weakening or dehiscence of capsulopalpebral fascia (lower lid retractor)
     ii. Degeneration of palpebral connective tissue separating the orbicularis muscle fibers and thus allowing preseptal fibers to override the pretarsal fibers
     iii. Horizontal laxity of the lid
5. **Mechanical entropion**
   - It occurs due to the lack of support provided by the globe to the lids
   - Therefore, it may occur in patients with phthisis bulbi, enophthalmos, and after enucleation or evisceration operation

B. Labeled Diagram of Lid

Cross Section of the Lid

Layers of the Eyelid (Fig. 2.1.1)

1. Skin
2. Subcutaneous tissue
3. Layer of muscle: Orbicularis oculi
4. Submuscular areolar tissue
5. Fibrous layer: Tarsal plate and orbital septum complex
6. A layer of non-striated muscle or Muller’s muscle
7. Conjunctiva

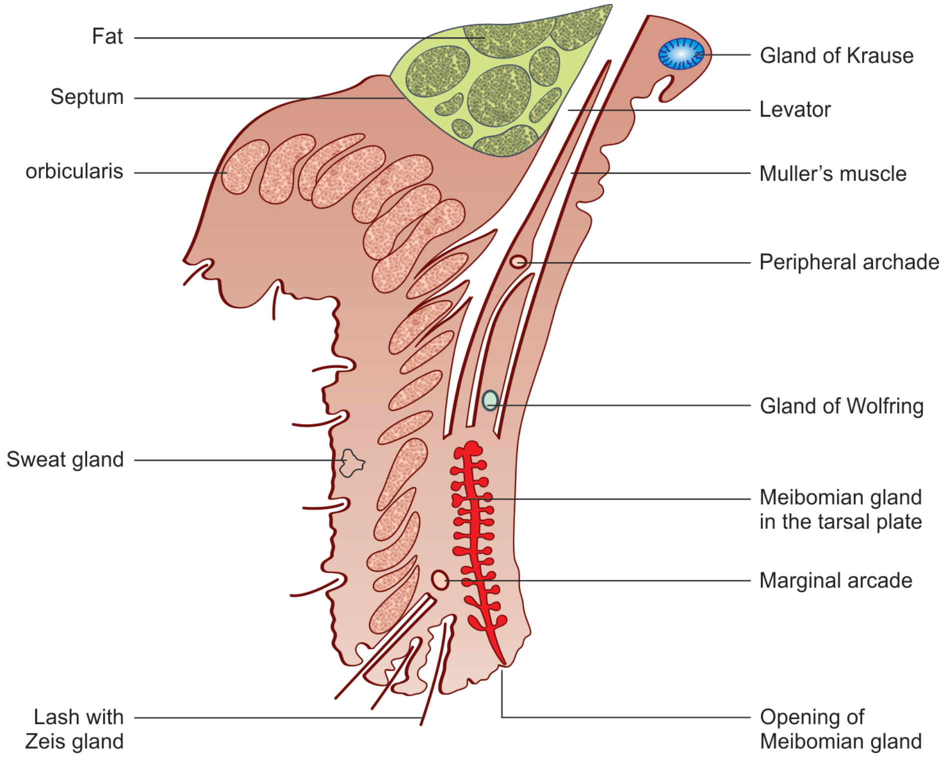

Fat
Septum
orbicularis
Sweat gland
Lash with
Zeis gland
Gland of Krause
Levator
Muller’s muscle
Peripheral archade
Gland of Wolfring
Meibomian gland
in the tarsal plate
Marginal arcade
Opening of
Meibomian gland

Fig. 2.1.1: Layers of the eyelid

Gland in the Eyelid

Glands of the eyelid	Location	Nature
Meibomian glands	Within the tarsal plate, around 30–40 in the upper lid and 10–20 in the lower lid	Modified sebaceous glands Responsible for outer lipid layer of the tear film
Glands of zeiss	Open into hair follicles	Sebaceous glands
Glands of moll	Open near the follicles, or into the ducts of glands of Zeiss	Modified sweat glands
Accessory lacrimal glands of Wolfring	Upper border of the tarsus	Responsible for basal secretion of tears

C. Sequelae of Entropion

Entropion causes inwards turning of eyelashes that in turn lead to recurrent corneal erosions.

D. Grading of Entropion

Grade I	In turning of the posterior lid margin Lash-globe contact on upgaze
Grade II	In turning of the inter marginal strip Lash-globe contact in primary gaze
Grade III	In turning of the anterior lid margin, gross lid distortion

E. Management

Depends on the etiology of entropion

1. Congenital Entropion

- May resolve with time
- May require plastic reconstruction of the lid
- After exposing the tarsal plate, adhesions are created between the tarsal plate and lower lid retractors and sutures are tightened till the lashes are turned outwards

2. Cicatricial Entropion

- *Anterior lamellar resection*
  - It is performed for mild entropion
  - Elliptical tissue of skin and orbicularis muscle are resected 3 mm away from the lid margin (Fig. 2.1.2)
- *Tarsal wedge resection*
  - It is done for moderate entropion, along with anterior lamellar resection, a wedge of tarsal plate is also resected (Fig. 2.1.3)
- *Kettsey's operation*
  - Done for severe entropion, in this surgery the distal tarsus is fractured and everted and sutured (Fig. 2.1.4)

3. Senile Entropion

- *Everting sutures*
  - Sutures are passed from the inferior fornix to emerge out from the lash line (Fig. 2.1.5)

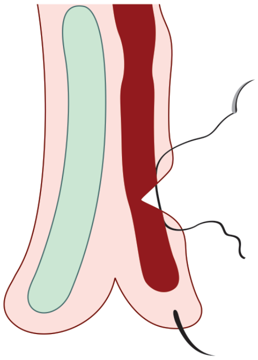


Fig. 2.1.2: Procedure of anterior lamellar resection

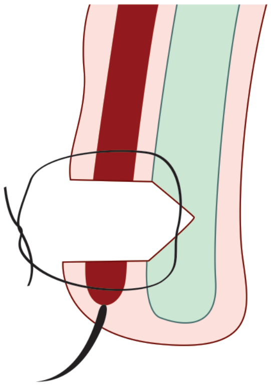


Fig. 2.1.3: Tarsal wedge resection

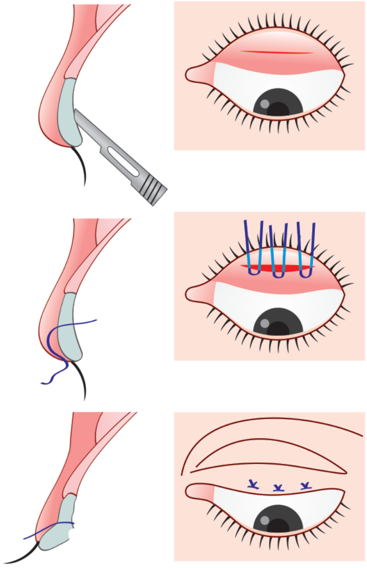


Fig. 2.1.4: Kettsey's operation

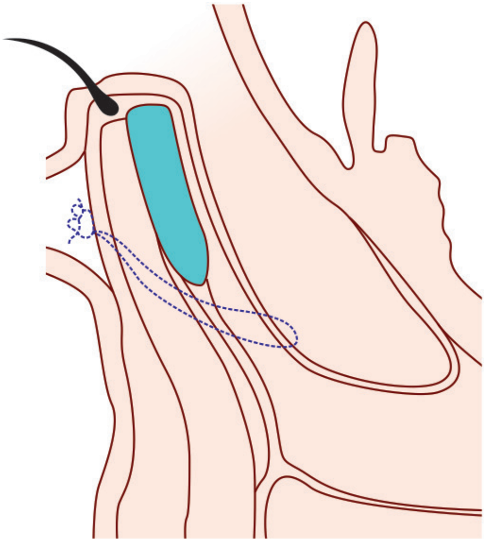


Fig. 2.1.5: Everting sutures

- This prevents overriding of the preseptal muscles
- This is a temporary procedure

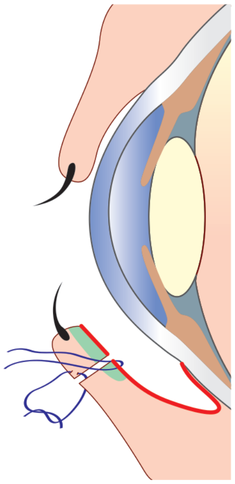


Fig. 2.1.6: Wies operation

- *Wies operation*
  - A horizontal incision is made 3 mm from the lower lid margin and mattress sutures are passed from the lower cut end of the tarsus to emerge at the lash line (Fig. 2.1.6)
- *Quickert procedure*
  - Indicated in marked lower lid laxity
  - Wies operation is combined with horizontal lid shortening

2. A 66-year-old woman presents with watering from the right eye for more than a year, which is continuously present with recurrent episodes of mucoid discharge. The discharge is controlled with topical eyedrops but tends to recur after the stoppage.

A. What is the most probable diagnosis? (1 mark)

B. Describe the clinical features of the same. (3 marks)

C. Describe the management of this case. (3 marks)

D. If the condition is seen in an infant, how does the management differ? (3 marks)

A. The Most Probable Diagnosis is

Right-sided chronic dacryocystitis

B. Clinical Features

The clinical presentation of dacryocystitis depends on the stage of the disease

Chronic catarrhal dacryocystitis	• Characterized by mild inflammation and obstructed flow of the tears • As a result, patients present with watering • On syringing: There is clear or mild mucoid regurgitation from the opposite punctum • The sac is usually healthy
Lacrimal mucocele	• Due to stagnation of tears, the sac distends • Patients present with watering or epiphora, which is more continuous than the previous stage and swelling in the sac area, regurgitation is positive with serous- mucoid discharge
Chronic suppurative dacryocystitis	• There is a pyogenic infection of the mucocele which now becomes a pyocele • Patients present with epiphora, recurrent conjunctivitis, and swelling at the inner canthus with mild erythema of the overlying skin • Regurgitation is positive with purulent discharge
Fibrotic sac	• Chronic inflammation of the sac leads to scarring • The sac loses its pump function and becomes atrophic

C. Management

- This case is probably in the first stage, as details about the regurgitation test are not mentioned
- The infection is controlled with topical antibiotics in the form of eyedrops and ointments
- Dacryocystorhinostomy is the preferred procedure as the sac is healthy:
  - This procedure is most physiological and eliminates epiphora
  - It is indicated when the sac and the nasal mucosa are healthy
  - If there is some nasal pathology like gross deviated septum or polyps, they must be first corrected as epiphora may subside by that alone
  - Endonasal DCR is another option
- Conventional external DCR
  - It is performed under general anesthesia in children and under sedation with local infiltration in adults
  - Before surgery, the nasal cavity is packed with ribbon gauze soaked in lignocaine + adrenaline
  - Skin is incised within 5 mm or more than 8 mm medial to medial canthus in a curvilinear fashion along the lacrimal crest
  - The subcutaneous tissue is bluntly dissected to reveal the medial palpebral ligament. It appears white. It is cut at the nasal end
  - Below the ligament, the sac is seen as pearly white. The sac is then dissected off the bone by blunt dissection
  - Sac is retracted laterally, and a bony window is created in the lacrimal bone of dimensions around 10–15 mm, and nasal mucosa is exposed; it is identifiable by its pink color
  - An ‘H” shaped flap is raised in the nasal mucosa. It is highly vascular and tends to bleed. Cotton soaked in adrenaline can be used to control bleeding

  - A similar flap is created on the lateral aspect of the sac. The corresponding flaps are sutured
  - MPL is sutured back to the periosteum and the subcutaneous tissue and skin are sutured
- Endonasal DCR
  - It is usually done with the help of an otorhinolaryngologist
  - The nasal cavity is prepared with local decongestants and local anesthesia
  - Conjunctiva is anesthetized with Paracaine and a 20 gauge illumination pipe is inserted into the upper punctum to sac to identify it
  - Endoscopically, a window is created in the nasal mucosa of the middle meatus, lacrimal bone either using instruments or using Ho-YAG laser
  - A similar incision is created in the sac and the fistula is intubated with silicone tubes
  - Postoperative intranasal steroids are given

D. Management of Watering in an Infant

- If there is watering of eyes in an infant, the most probable cause is congenital nasolacrimal obstruction
- The management depends on the age of the child

Crigler massage	
Age <1 month	The mother is instructed to massage over the lacrimal sac area with the help of her little finger during every feed, the direction of the massage should be towards the ala of the nose This is supported by topical antibiotics
Up to 2 months	Lacrimal syringing with saline is done once a week. Hydraulic pressure is supposed to break membranous openings, if any
3–4 months	If epiphora continues, probing the nasolacrimal duct should be tried under general anesthesia. This is done using a Bowman probe, passing through one of the puncta, across the canaliculi, sac, and to the nasolacrimal duct
After 6 months	Silicone tube intubation in the nasolacrimal duct for 6 months Later in life, DCR is the procedure of choice

3. 56-year-old presents with left-sided drooping of his upper eyelid noticed for 5 days. He has no other visual or ocular symptoms. He is a chronic smoker for 40 years and has been having a cough, associated with weight loss. On examination, there is mild ptosis, left-sided miosis, and an excellent levator function. Extraocular movements are normal.

A. State the probable cause of ptosis in this patient. (1 mark)

B. Describe the oculosympathetic pathway with a neat diagram. (4 marks)

C. Classify ptosis based on etiology. (4 marks)

D. Enumerate two causes of neurogenic ptosis. (1 mark)

A. Probable Cause of Ptosis

Given the history of ptosis in a man with a history of smoking, cough, and weight loss, Horner's syndrome is probable due to Pancoast's tumor (Carcinoma of the apex of the lung) that compresses the superior cervical ganglion.

B. Oculosympathetic Pathway (Fig. 2.1.7)

Stimulation of this pathway leads to mydriasis and lid retraction

Posterior hypothalamus

↓

Descends along the brainstem

↓

Ciliospinal center of Budge, between C8 and T2

↓

Preganglionic fibers traverse along apical pleura and synapse at the superior cervical ganglion

↓

Postganglionic fibers again enter the cranial cavity by ascending close to internal carotid and travel within the substance of the cavernous sinus

↓

Ophthalmic division of trigeminal nerve

↓

Ciliary ganglion (but does not relay here)

↓

Long ciliary nerves

↓

Dilator pupillae, Muller's muscle

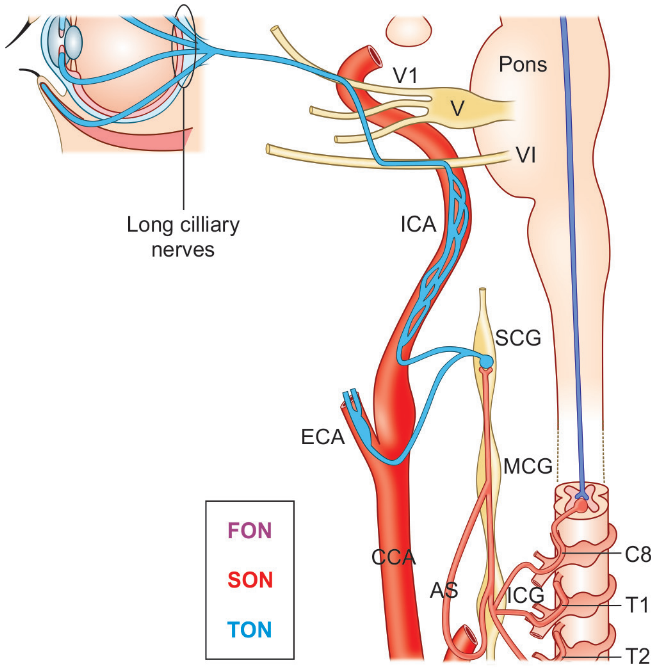
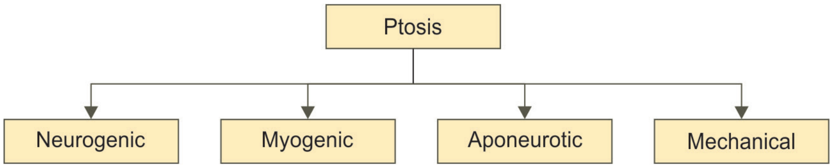

V1
Pons
V
VI
Long cilliary nerves
ICA
SCG
ECA
MCG
FON
SON
TON
CCA
C8
AS
ICG
T1
T2

Fig. 2.1.7: Oculosympathetic pathway

C. Classification of Ptosis

- It can be congenital or acquired
- Etiologically it is classified into four types



D. Causes of Neurogenic Ptosis

1. Horner's syndrome
2. Third nerve palsy

SHORT ESSAYS

1. A 40-year-old male presents with a pricking sensation of the left eye. On examination, the eyelashes in the lower lid were found to be turned inwards.

A. Identify the diagnosis (1 marks)
B. Describe the etiology and clinical features of the same. (3 marks)
C. Describe the management of the same. (2 marks)

A. Diagnosis

This is a case of trichiasis.

B. Etiology and Clinical Features

Etiology	Clinical features
• Cicatrizing trachoma • Ulcerative blepharitis • Healed membranous conjunctivitis • Chemical and mechanical injuries • Burns • An operative scar on the lid margin	• Foreign body sensation • Lacrimation • Photophobia • Pain

C. Management

- Epilation: Removal of the trichiatic lashes, but trichiasis recurs when the lashes grow back
- Destruction of the follicle by electrolysis and cryoepilation
- Surgical correction for entropion
- Supportive therapy in the form of tear film substitutes and antibiotics. Temporarily, a tape can be applied by retracting the lower lid such that the lid is everted

2. Classify blepharitis. Describe the clinical features of each type. (1+4 marks)

Blepharitis

Classification

Classified as anterior and posterior blepharitis depending on the lamella of the eyelid that is involved

A. *Anterior blepharitis*
   1. Seborrheic blepharitis
   2. Bacterial or staphylococcal blepharitis
   3. Parasitic blepharitis

B. *Posterior blepharitis/meibomitis*

Clinical Features

Type	Features	
Seborrheic blepharitis	Symptoms	• Itching and ocular irritation • Patients may notice whitish material on lid margins • Loss of lashes occasionally
	Signs	• White dandruff-like scales along the lash line with an underlying hyperemic surface • Thickened lid margins • May be associated with dry eye
Bacterial blepharitis	Symptoms	• Ocular irritation and itching of chronic nature • More pronounced in the morning • Gluing of cilia, loss of cilia
	Signs	• Yellow crusts at the base of the lashes that glue the lashes together • On removal of these lashes, small ulcers are seen • Underlying dilated blood vessels
Parasitic blepharitis (phthiriasis palpebrum)	Symptoms	• Chronic ocular irritation, itching, burning sensation
	Signs	• Lid margins are thickened and inflamed • Crab lice are seen anchoring into the lid margins • Nits or eggs are seen as gray pearl-like structures • Conjunctiva is congested and shows a follicular reaction
Posterior blepharitis/ meibomitis	Symptoms	• Usually chronic • Presents with features of dry eyes like grittiness, itching and tends to occur in a recurring fashion
	Signs	• Foamy discharge is seen at the canthi and lid margins • The opening of the Meibomian glands shows prominent and thick secretions that can be expressed. • There are dilated vessels surrounding the Meibomian openings • On eversion of the lids, yellowish streaks are seen vertically along the length of the Meibomian glands

3. Describe the management of chalazion. (5 marks)

Chalazion

- A chronic granulomatous inflammation of the Meibomian gland leading to occlusion of its opening and collection of the secretion to form a Meibomian cyst
- It is asymptomatic and its removal is indicated only if the chalazion is big, bothersome to the patient, and if malignancy is suspected (as in older age of presentation, lid margin distortion)

- The options for management are conservative and surgical
  1. Conservative management includes warm compresses over the chalazion with a massage
  2. Incision and curettage (I and C) (Fig. 2.1.8)
     - Standard of care of treatment of chalazion.
     - The chalazion is identified on the conjunctival side by a yellow-gray appearance
     - Then, that part of the lid is anesthetized with lignocaine infiltration
     - A chalazion clamp is placed and tightened
     - The lid is everted
     - A No. 11 blade is used to make a vertical incision along the length of the lid and the yellow creamy material is removed
     - A scoop is used to scoop out the contents and break any loculations in the chalazion. The clamp is removed, and the eye is padded
     - If the chalazion must be curetted from the skin side, a horizontal incision is made
  3. Intralesional injection of Triamcinolone
     - Used if incision and curettage is difficult as in if the chalazion is located close to the punctum or is too small
     - In this method, after instilling topical proparacaine, a 27-gauge needle is used to inject the below-mentioned dose of the drug into the center of the chalazion
     - The dose depends on the size of the chalazion
     - The patient is instructed to continue giving warm compresses

<1 cm	2 mg/0.05 ml
1-1.5 cm	4 mg/0.1 ml
>1.5 cm	6 mg/0.15 ml

  4. Diathermy: It is indicated for marginal chalazion
  5. Oral tetracycline is indicated for individuals with multiple chalazia and associated acne rosacea

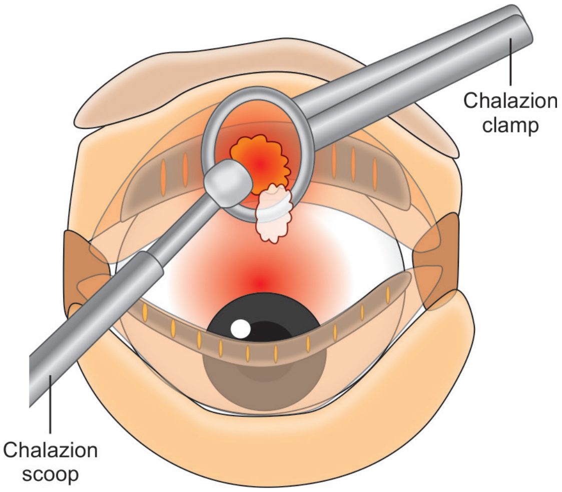


Fig. 2.1.8: Method of chalazion: I and C

4. A 16-year-old college-going girl presents with pain and swelling in the upper lid of the right eye. On examination, there is lid edema, erythema, and a pus point are found posterior to the gray line.

A. State the most probable diagnosis.	**(1 mark)**
B. Enumerate four causes for recurrence of this inflammation.	**(2 marks)**
C. Describe the management of this case.	**(2 marks)**

A. Most Probable Diagnosis

Internal hordeolum of the right upper lid.

B. Causes of Recurrent Internal Hordeola

1. Uncorrected refractive error especially hypermetropia
2. Uncorrected phorias
3. Diabetes mellitus
4. Nasal carriage of *Staphylococcus*

C. Management

- Since there is a pus point seen, this is the stage of abscess formation
- The pus is drained under topical anesthesia by puncturing the abscess with a 26-gauge needle
- The area is massaged till all the pus is out and there is sanguineous discharge

5. A 6-year-old boy is brought to the outpatient department from an orphanage with a history of multiple swellings over the upper lid margins in both eyes. On examination, there are multiple swellings of around 4 mm diameter along the lid margin and skin of the upper lids and they have a central umbilication.

A. What is the most likely diagnosis?	**(1 mark)**
B. Describe the etiology and sequelae of this condition.	**(2 marks)**
C. Describe the treatment of this condition.	**(2 marks)**

A. Most Likely Diagnosis

Molluscum contagiosum.

B. Etiology and Sequelae

Etiology	Sequelae
• Viral in nature • Caused by poxvirus • Multiple mollusca are seen in immunosuppressed conditions	• Follicular conjunctivitis due to the irritation of the conjunctiva caused by the material within the lesion • Superficial keratitis

C. Management

- They are contagious
- The lesions should be incised, and the interior is cauterized with a tincture of iodine or carbolic acid

6. A 28-year-old lady presents with a painless swelling in the right upper lid for 3 weeks. On examination, a firm, non-tender, and well-circumscribed mass is felt over the tarsal plate, 5 mm in diameter. On everting the eyelid, there is a greyish appearance of the overlying conjunctiva.

A. What is the most probable diagnosis? (1 mark)

B. Name one cause for recurrence of this swelling even after complete removal. (1 mark)

C. Describe the etiology and methods of treatment of the condition. (3 marks)

A. Most Probable Diagnosis

Chalazion of the right upper lid

B. Cause of Recurrence

Malignancy of the meibomian gland

C. Etiology and Methods of Treatment

Etiology

- Uncorrected refractive errors
- Partially treated hordeolum internum
- Acne rosaceae
- Meibomitis
- Diabetes

Methods of Treatment

- Warm compresses
- Incision and curettage
- Intralesional triamcinolone
- Diathermy
- Oral tetracycline

7. Describe the causes, grading, and clinical features of ectropion. (2+1+2=5 marks)

Ectropion

Causes

1. **Senile ectropion**
   - It is the most common variety and involves only the lower lids, and occurs due to senile laxity of the tissues
2. **Cicatricial ectropion**
   - Due to scarring of the skin due to thermal burns, chemical burns, lacerating injuries, scleroderma, radiation-induced tight skin, and skin ulcers
3. **Paralytic ectropion**
   - It is due to facial paralysis and thus affects only the lower lids
   - Facial palsy can be due to Bell's palsy, head injury, parotid tumors, and infections of the middle ear
4. **Mechanical ectropion**
   - Due to mechanical traction over the lower lid or due to severe proptosis

Grading

Grade I	Only punctum is everted
Grade II	The lid margin is everted, and palpebral conjunctiva is visible
Grade III	The fornix is also visible

Clinical Features

Symptoms

- Watering or epiphora
- Irritation of the eye, discomfort

Signs

- Outrolling of the lid margin
- Skin scar or features of 7th nerve palsy may be visible
- Horizontal lid laxity can be demonstrated by snap test
- Medial canthal laxity: Movement of the inferior punctum >2 mm on gentle traction on the lower lid
- Lateral canthal tendon laxity

8. Describe the various procedures for the treatment of ectropion. (5 marks)

Treatment of Ectropion

1. **Senile ectropion**
    - Lateral tarsal strip procedure: It is done for lateral canthal tendon laxity and the lateral tarsal strip is excised and attached to the lateral orbital rim
    - Medial conjunctivoplasty: It is indicated in presence of medial canthal tendon laxity. Diamond-shaped conjunctiva and subconjunctival tissue are excised
    - Byron Smith's modified Kuhnt–Szymanowski operation: It is reserved for severe ectropion in which a pentagonal full-thickness excision from the lateral third of the eyelid is combined with triangular excision of the skin from the area just lateral to lateral canthus to elevate the lid (Fig. 2.1.9)

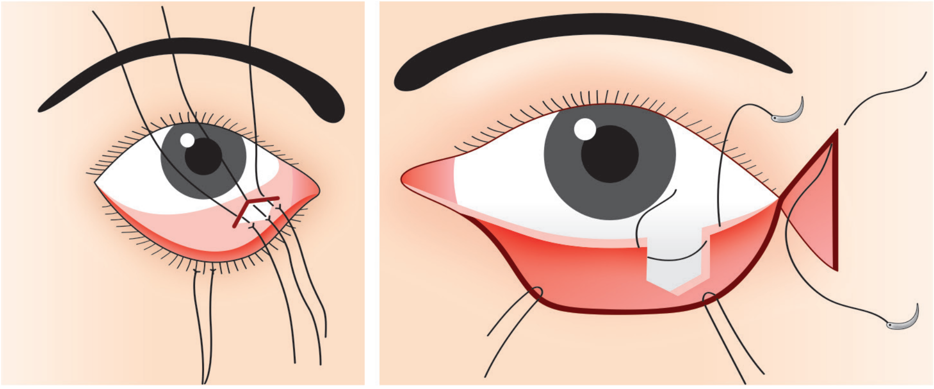


Fig. 2.1.9: Byron Smith's method

2. **Paralytic ectropion**
    - Lateral tarsorrhaphy or palpebral sling operation, in which a fascia lata sling is passed in the subcutaneous layer all around the lid margins (Fig. 2.1.10)
3. **Cicatricial ectropion**
    - V-Y operation—for mild ectropion: In it a V-shaped incision is given, the skin is undermined and sutured in a Y-shaped pattern (Fig. 2.1.11)
    - Z-plasty (Elschnig's operation): Moderate degree of ectropion
    - Excision of scar tissue and full-thickness skin grafting is done in severe cases. A skin graft may be taken from the upper lid, behind the ear, or the inner side of the upper arm
4. **Mechanical eectropion**
    - It is corrected by treating the underlying cause

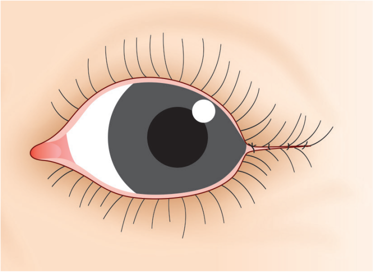


Fig. 2.1.10: Lateral tarsorrhaphy

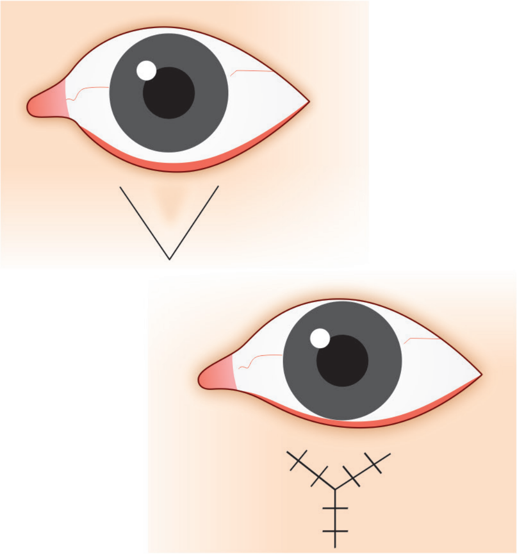


Fig. 2.1.11: V–Y operation

9. Describe the etiology, clinical features, and management of preseptal cellulitis. (2+1+2 marks)

Preseptal Cellulitis

Etiology

- Preseptal cellulitis is the inflammation of the soft tissue anterior to the orbital septum
- It is usually caused by *Staphylococcus aureus* or *Streptococcus pyogenes*
- Commonly an internal or external hordeolum spreads to the preseptal space. Other ways are through injury to lids and infection, acute dacryocystitis

Clinical Features

- Lid edema and erythema associated with pain
- Source of infection may be evident on inspection (hordeola, dacryocystitis, etc.)
- The rest of the ocular examination is normal (visual acuity, extraocular movements)

Management

- Topical antibiotics
- Systemic anti-inflammatory drugs
- Systemic antibiotics like co-amoxiclav or cloxacillin
- Surgical exploration and debridement may be required in presence of an abscess

10. Describe 5 tests in investigating a case of dacryocystitis. (5 marks)

1. **Fluorescein dye disappearance test**
    - In this test, a drop of 2% fluorescein is instilled in the conjunctival sac
    - After 2 minutes the eyes are examined for the presence of fluorescein

- Normally, no dye is present after 2 minutes
- Retention of dye in the conjunctival sac after 2 minutes is suggestive of insufficient drainage

2. **Probing and syringing**
   - In this test, after instilling topical anesthesia and dilating the punctum, a Bowman's probe is passed through the punctum (Fig. 2.1.12)
   - If a hard stop is felt (due to the probe touching the bone), there is no canalicular obstruction
   - If a soft stop is felt, there is canalicular obstruction
   - At the same time, 2 ml of normal saline is gently passed through the puncta with a 26- or 30-gauge cannula

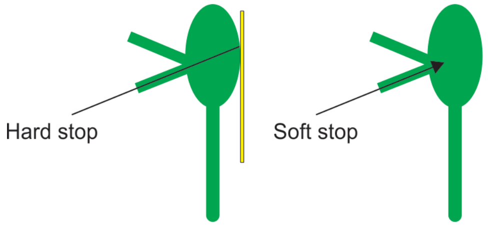


Fig. 2.1.12: Results of probing

Regurgitation from same punctum	Obstruction is within the canaliculus or junction of punctum with common canaliculus
Regurgitation from opposite punctum	Obstruction is distal to canaliculus (common canaliculus or sac or nasolacrimal duct)
Partial regurgitation from opposite punctum, with the patient feeling salty taste in the throat	Partial obstruction
Free flow, patient feeling salty taste in the throat	Epiphora is probably due to pump failure, no obstruction

3. **Jones dye test**
   - It is an extension of the fluorescein dye disappearance test
   - It is used to locate the location of the block in partial obstruction
   - Primary jones test
     - Used to differentiate primary hypersecretion from partial obstruction
     - In this test, fluorescein is instilled in the conjunctival sac, and a cotton-tipped bud soaked in local anesthetic is placed under the inferior turbinate
     - Positive: Fluorescein recovered from the nasal cavity indicates primary hypersecretion
     - Negative: No fluorescein in nasal cavity—suggestive of obstruction, or lacrimal pump failure
   - Proceed to secondary Jones test
   - Secondary Jones test
     - In this step, residual fluorescein is washed from the sac. Saline is injected into the canaliculi
     - Positive: Fluorescein recovered from the nasal cavity suggests that dye had primarily entered the sac but could not reach the nasal cavity
     - Negative: Dye is not recovered from the nasal cavity. Dye had not primarily entered the lacrimal sac. It may be due to canalicular obstruction or due to lacrimal pump failure
4. **Dacryocystography**
   - A radio-opaque dye is injected into the lower punctum in both eyes using a catheter and radiographs are taken, after ten minutes, the patient is made to stand and again radiographs are taken to study the effect of gravity on drainage
   - It can detect the site of obstruction

5. **Radionucleotide scintigraphy**
   - The principle is similar to dacryocystography, but radionucleotide technetium-99 is used to inject into the canaliculus, and a gamma camera is used to take photographs

11. A patient has a malignant tumor of the parotid gland. State the ocular condition that occurs following resection of the parotid gland leading to damage of a particular cranial nerve. Enumerate the treatment options for the same? (1+4=5 marks)

- The facial nerve passes through the parotid gland
- After resection of a malignant parotid tumor, the facial nerve is affected
- As a result, there is permanent facial palsy leading to lagophthalmos

Management

- Temporary tarsorrhaphy—creating appositions between the upper and lower lid to protect the cornea is done by suturing the lids up to half the corneal surface and is done on the lateral surface. In paramedian tarsorrhaphy, lids are sutured on either side of the pupil
- Permanent tarsorrhaphy is done when there is no hope of facial recovery. There are raw surfaces created by removing a strip of mucosa and then suturing
- Gold weights can be implanted within the lids to bring the lids down
- Supportive measures are lubricants and lid taping. Lubricants are given in the form of eyedrops and gel to keep the ocular surface moist. Lid taping is advised when the patient retires to bed

12. Describe the grading of ptosis. Explain Berke's method of measuring levator excursion. (2+3 marks)

Grading of Ptosis

Mild ptosis	<2 mm
Moderate ptosis	2–4 mm
Severe ptosis	>4 mm

Berke's Method

- The patient is instructed to look in extreme downgaze, and the zero mark of the ruler is placed adjacent to the central upper lid margin
- The frontalis muscle is fixated with direct pressure applied to the eyebrow and the patient is instructed to look in extreme upgaze (Fig. 2.1.13)
- The number of millimeters on the ruler gives the excursion of the upper eyelid i.e., levator function. Normally, it is around 15 mm

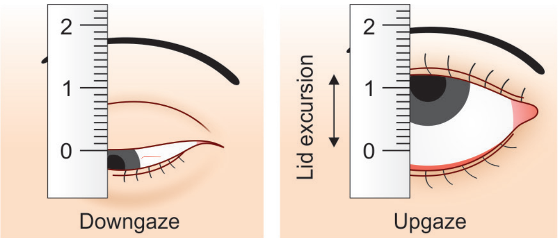


Fig. 2.1.13: Berke's method
≥15 mm: Normal, 12–14 mm: Good,
5–11 mm: Fair, ≤4 mm: Poor

13. Explain the surgical correction of moderate ptosis in a patient with sub-normal levator function. (5 marks)

When the levator function is sub-normal, either levator resection or frontalis suspension surgeries can be performed

Levator Resection

- External or transcutaneous is most commonly used when levator function is normal and the upper lid crease is high
- In this setting, the levator muscle is normal, but the aponeurosis is stretched or disinserted
- Depending on the levator function assessed by Berke's method, the amount of resection is decided

Levator function	Amount of levator to be resected
<10 mm	10–13 mm
5–10 mm	12–17 mm
<4 mm	18–25 mm

- In the conjunctival approach, (Blasckovics operation), the palpebral conjunctiva is incised to expose the tarsal border
- The levator horns are exposed and the aponeurosis is excised

Frontalis Sling Surgery

- The eyelid-elevation is enhanced by the frontalis muscle normally lifts the brow and contributes to eyelid elevation is enhanced by connecting the frontalis muscle and eyebrow to the eyelid with a subcutaneous sling
- Indications are severe ptosis with moderate to poor levator function. Also, ptosis associated with aberrant regeneration, and Marcus Gunn's ptosis is benefited from this

SHORT ANSWERS

1. A 35-year-old woman presents with target lesions on skin and blisters with mucosal erosions following ingestion of phenytoin. Enumerate the sequelae of ocular features of this disease? (3 marks)

- The dermatological features following ingestion of phenytoin point to a diagnosis of Stevens–Johnson syndrome (SJS)
- Phenytoin is a known trigger for a drug reaction
- SJS is a hypersensitivity disorder that follows a reaction to a drug or infection by an organism, that leads to erosion of skin and mucous membranes

Sequelae of SJS in eye

- The conjunctiva is also affected. Erosions are formed on the conjunctival surface that ulcerate
- Also, they form adhesions when the raw surfaces are opposed for a long time
- This leads to the formation of symblepharon
- Also, due to inflammation, there is damage to the tarsal plate
- There is in turning of the tarsal plate leading to cicatricial entropion. As a result, there is trichiasis, which causes recurrent corneal erosions
- The Goblet cells of the conjunctiva are damaged in the inflammatory process. As a result, there is mucin-deficiency dry eye

- A dry eye further accelerates the corneal erosions
- Also, there is damage to the limbal stem cells. If there is extensive inflammation, total limbal stem cell deficiency can result

2. Describe the clinical presentation of staphylococcal blepharitis. (3 marks)

Symptoms	Signs
• Chronic irritation in the eyes • Itching • On and off epiphora • Photophobia • Loss of lashes	• Yellow crusts are seen at the root of lashes • When the crusts are gently removed, small ulcers are seen to bleed • Thickening of lid margins dilated blood vessels along the lid margins • Conjunctival congestion may be present

3. Define

A. Symblepharon (1 mark)
B. Ankyloblepharon (1 mark)
C. Blepharophimosis (1 mark)

A. Symblepharon

Adhesions between palpebral and bulbar conjunctiva

B. Ankyloblepharon

Adhesion between upper and lower lid margins

C. Blepharophimosis

A condition in which the horizontal width of the lids is smaller than expected

4. Define the terms

A. Tylosis (1 mark)
B. Poliosis (1 mark)
C. Madarosis (1 mark)

A. Tylosis

Thickening of the lid margins due to thickened tarsal margins

B. Poliosis

Abnormal whitening or greying of cilia of the eyelids

C. Madarosis

Loss of eyelashes or eyebrows

- Supraciliary madarosis refers to the loss of hair from eyebrows
- Ciliary madarosis refers to the loss of cilia of eyelids

5. Explain the management of phthiriasis palpebrarum. (3 marks)

Management of Phthiriasis Palpebrarum

- Phthiriasis palpebrarum is infestation of the eyelid with crab lice
- Mechanical removal of nits and adult crab lice
- Usually, the crab lice tightly adhere to the lashes. 1% pilocarpine is applied over the lids. This causes paralysis of the lice and then they can be removed easily

- Other topical options for application are petroleum ointments, 20% fluorescein, and yellow mercuric oxide ointment
- Some have reported good results with cryotherapy and argon laser photocoagulation
- Oral Ivermectin is given
- Dermatological evaluation is required for treatment of pubic lice and psychological assessment to rule out sexual abuse in relevant cases may be required

6. Describe the frontalis sling surgeries. (3 marks)

- In moderate or severe ptosis with poor levator function, a frontalis sling is advised
- Eyelid elevation is enhanced by the frontalis muscle normally lifts the brow and contributes to eyelid elevation is enhanced by connecting the frontalis muscle and eyebrow to the eyelid with a subcutaneous sling
- Double rhomboid frontalis sling with supramidal suture (Fig. 2.1.14)

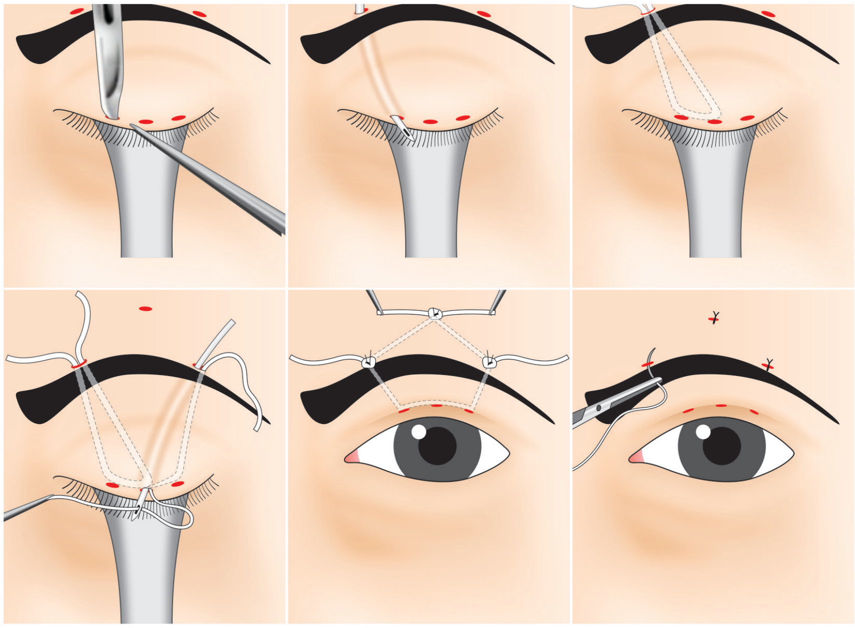


Fig. 2.1.14: Frontalis sling—duble rhomboid suture

  - In this technique, supramid suture is used to make a double rhomboid shaped sling
- Crawford's sling with triangular placement (Fig. 2.1.15)
  - In this technique, fascia lata is used
  - Three incisions are made as done previously, except that the central brow incision is 1cm higher
  - The slings made from fascia lata are used to tie in a pentagon shape as shown in the picture

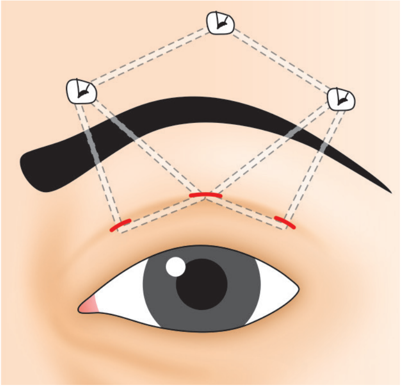


Fig. 2.1.15: Crawford's technique

7. Explain the Marcus Gunn jaw winking phenomenon and its importance in a patient with ptosis. (3 marks)

Marcus Gunn Jaw Winking

- A phenomenon that is usually seen in congenital myogenic ptosis
- In presence of this, the ptosis is termed as 'complicated' ptosis
- There is uplifting of the eyelid when the jaw opens, or the lateral pterygoid is put into action
- Other movements that cause upshoot of the eyelid are chewing, jaw movement to the opposite side, jaw protrusion, swallowing, and clenching
- This occurs due to aberrant innervation between the nerves supplying the muscles of mastication and the superior branch of the oculomotor nerve

Importance

- Simple myogenic congenital ptosis is usually successfully corrected by Levator resection
- However, in presence of Marcus Gunn jaw winking phenomenon, there is worsening of the lid upshoot following levator resection
- These patients are best treated by sling surgeries

8. Describe the surgery for correction of ptosis due to Horner syndrome. (3 marks)

- The surgery advocated for mild ptosis due to Horner syndrome is Fasanella Servat surgery or conjunctivo—mullerectomy (Fig. 2.1.16)
- The lid is anesthetized by infiltration of lignocaine given in the subconjunctival space
- The lid is everted by Desmarre's retractor
- Not more than 3 mm of the tarsal plate is marked with a surgical marker
- Two hemostats are placed just above the marked line to grasp the tarsal plate
- Skin and the orbicularis should be free from the clamp
- Horizontal mattress sutures are placed below the clamp and No.15 blade is used to excise the upper edge of the tarsus and the Muller muscle and the conjunctiva

9. Give the anatomical basis for lacrimal pump mechanism. (3 marks)

- The lacrimal pump is a physiological mechanical pump that helps in drainage of tears from the lacus lacrimalis (tear lake at the medial canthus)
- When the lids close, the preseptal orbicularis oculi contract and close the puncta and the canaliculi. At the same time, the fibers of preseptal orbicularis that are inserted into the lacrimal sac open the sac
- The tears are drained towards the lacrimal sac due to the negative pressure created

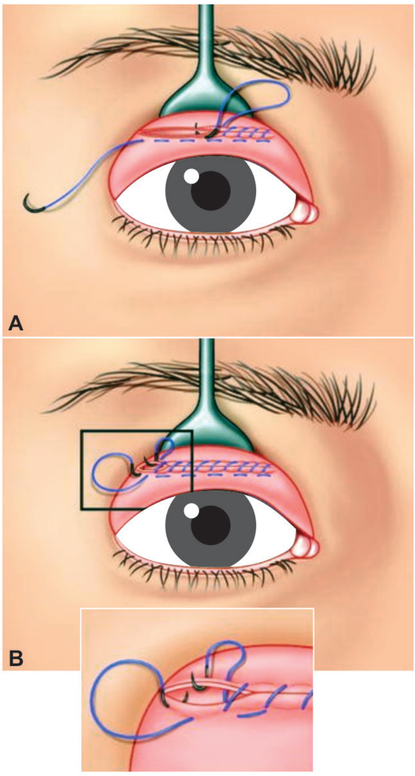


Fig. 2.1.16: Conjunctivo-Mullerectomy

- When the lids open, the muscle relaxes, and the puncta and canaliculi open causing them to refill from the lacus lacrimalis (Fig. 2.1.17)

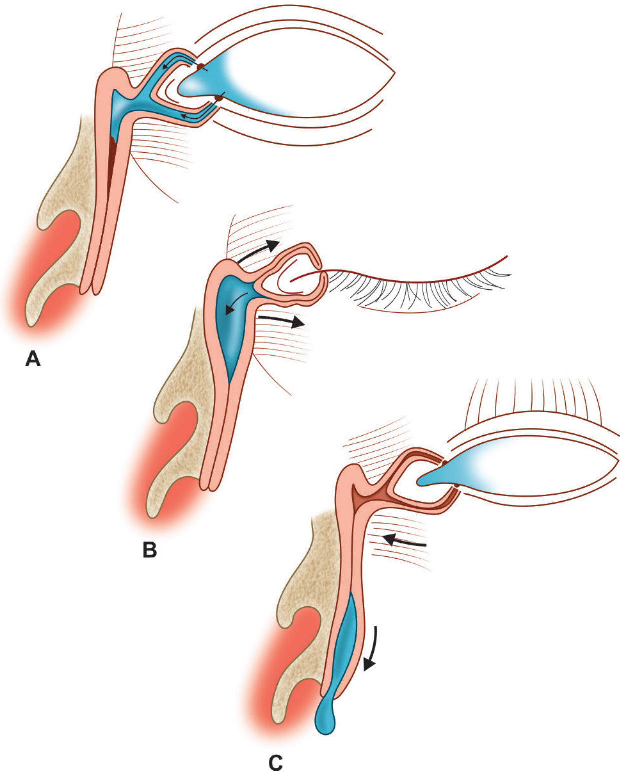


Fig. 2.1.17: Lacrimal pump mechanism

COMPETENCY

OP2.2 DEMONSTRATE THE SYMPTOMS AND CLINICAL SIGNS OF CONDITIONS ENUMERATED IN OP2.1

SHORT ESSAYS

1. Enumerate four tests used in the evaluation of a watering eye. Elaborate on the procedure and implications of performing Jones tests. (2+3 marks)

Tests Used in the Evaluation of a Watering Eye

1. Fluorescein dye disappearance test
2. Probing and syringing
3. Jones dye test
4. Dacryocystography

Jones Dye Test

- It is an extension of the fluorescein dye disappearance test
- It is used to locate the location of the block in partial obstruction
- Primary Jones test
  - Used to differentiate primary hypersecretion from partial obstruction
  - In this test, fluorescein is instilled in the conjunctival sac, and a cotton-tipped bud soaked in local anesthetic is placed under the inferior turbinate

  - Positive: Fluorescein recovered from the nasal cavity indicates primary hypersecretion
  - Negative: No fluorescein in nasal cavity → suggestive of obstruction, or lacrimal pump failure
- Proceed to secondary Jones test
- Secondary Jones test
  - In this step, residual fluorescein is washed from the sac. Saline is injected into the canaliculi
  - Positive: Fluorescein recovered from the nasal cavity suggests that dye had primarily entered the sac but could not reach the nasal cavity
  - Negative: Dye is not recovered from the nasal cavity. Dye had not primarily entered the lacrimal sac. It may be due to canalicular obstruction or due to lacrimal pump failure

2. Enumerate various measurements in ptosis evaluation. Explain the levator excursion measurement. (3+2 marks)

Ptosis Measurements

1. Palpebral fissure height (PFH)
2. Marginal reflex distance 1
3. Marginal reflex distance 2
4. Levator excursion
5. Height of eyelid crease
6. Corneal sensation

Levator Excursion Measurement (Fig. 2.2.1)

- The patient is instructed to look in extreme downgaze, and the zero marks of the ruler is placed adjacent to the central upper lid margin
- The frontalis muscle is fixated with direct pressure applied to the eyebrow and the patient is instructed to look in extreme upgaze
- The number of millimeter on the ruler gives the excursion of the upper eyelid, i.e., levator function. Normally, it is around 15 mm

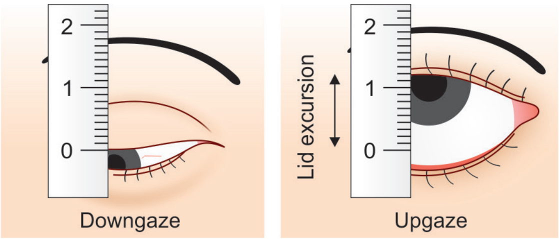


Fig. 2.2.1: Berke's method
≥15 mm: Normal, 12–14 mm: Good,
5–11 mm: Fair, ≤4 mm: Poor

SHORT ANSWERS

1. Explain the clinical features of the dermoid. (3 marks)

- Dermoid arises due to pinching off of a few epithelial precursors from the neurectoderm into the subcutaneous tissue during development
- The cyst lining resembles skin with keratinizing epithelium with hair follicles
- Patients usually present in their second or third decade with painless, non-progressive swelling in the lateral or medial aspect of their brow
- The typical dermoid appears firm, well encapsulated, localized lesion in the superotemporal or superonasal quadrant
- It is freely mobile and is not attached to the underlying bone. The skin is pinchable over the swelling
- Sometimes, dermoids can arise from within the orbit and thus can lead to bony defects and eccentric proptosis
- Complete surgical excision is the treatment of choice

2. Identify the procedure and explain it. (1+2 marks)

The name of the procedure is lacrimal sac syringing.

Procedure (Fig. 2.2.2)

- The patient is made to lie in the supine position
- A topical anesthetic is instilled in both eyes
- A 2 cc syringe is filled with normal saline and is attached with a 26 guage cannula
- The lower lid of the patient is mildly everted to expose the lower punctum
- The cannula is passed into the punctum and then turned medially to move in the direction of the canaliculus
- Now, the saline is pushed
  - If there is no block, the patient feels the salty taste in the throat as the saline trickles down the nasal cavity into the nasopharynx and oropharynx
  - If there is a block, saline regurgitates from the opposite or the same punctum (depending on the level of block)
- The procedure is repeated on the other side

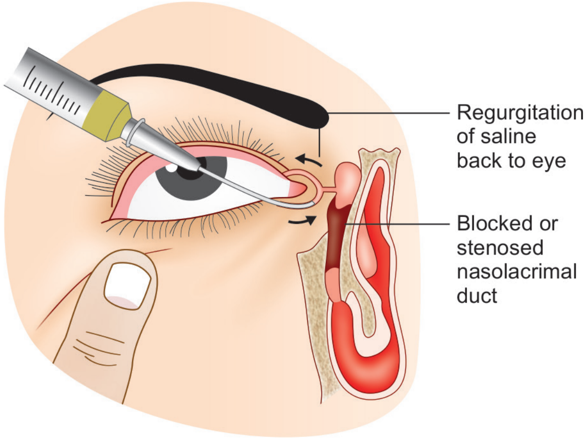

Regurgitation of saline back to eye
Blocked or stenosed nasolacrimal duct

Fig. 2.2.2: Procedure of lacrimal syringing

Inferences

Regurgitation from same punctum	Obstruction is within the canaliculus or junction of punctum with common canaliculus
Regurgitation from opposite punctum	Obstruction is distal to canaliculus (common canaliculus or sac or nasolacrimal duct)
Partial regurgitation from opposite punctum, with the patient feeling salty taste in the throat	Partial obstruction
Free flow, patient feeling salty taste in the throat	Epiphora is probably due to pump failure
No obstruction	

3. Describe the procedure of demonstrating

A. Horizontal lid laxity (1 mark)
B. Medial canthal tendon laxity (1 mark)
C. Lateral canthal tendon laxity (1 mark)

A. Horizontal Lid Laxity

- It is demonstrated by the 'snap test'. In the test, the middle of the lower lid is pinched off the globe and let go
- The lid fails to snap back to its position of opposing the globe
- It is seen in the weakness of orbicularis and senile ectropion

B. Medial Canthal Tendon Laxity

- It is demonstrated by applying gentle traction on the lower lid near the medial canthus
- The movement of the inferior punctum >2 mm from its normal position is positive for medial canthal tendon laxity

C. Lateral Canthal Tendon Laxity

- There is a rounded appearance of the lateral canthus and it moves more than 2 mm when pulled laterally

COMPETENCY

OP2.3 DEMONSTRATE UNDER SUPERVISION CLINICAL PROCEDURES PERFORMED IN THE LID INCLUDING: BELL'S PHENOMENON, ASSESSMENT OF ENTROPION/ECTROPION, PERFORM THE REGURGITATION TEST OF LACRIMAL SAC. MASSAGE TECHNIQUE IN CONG. DACRYOCYSTITIS, AND TRICHIATIC CILIA REMOVAL BY EPILATION

SHORT ANSWERS

1. Demonstrate the understanding of the procedure of lacrimal probing. (3 marks)

- In this test, after instilling topical anesthesia and dilating the punctum, a Bowman's probe is passed through the punctum
- The probe is passed vertically in the direction of the canaliculus and then turned medially towards the lacrimal fossa
- This test is used to differentiate between canalicular obstruction and post-canalicular obstruction (Fig. 2.3.1)
  - If a hard stop is felt (due to the probe touching the bone), there is no canalicular obstruction
  - If a soft stop is felt, there is canalicular obstruction

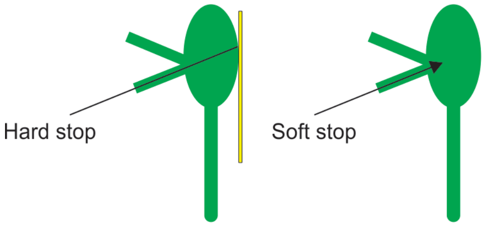


Fig. 2.3.1: Results of lacrimal probing

2. Explain how the regurgitation test is performed and its interpretation. (3 marks)

- This test is based on the principle that, if there is an obstruction distal to the lacrimal sac area, the tears and other secretions get stagnated
- In this test, firm gentle pressure is applied over the lacrimal sac area
- In cases of obstruction, there is a regurgitation of clear or mucoid material depending on the stage of the dacryocystitis
- Possible results from regurgitation test

No regurgitation, no swelling	No obstruction
No regurgitation, but the swelling is present over the lacrimal sac	Encysted mucocele
Clear regurgitation from either punctum	Obstruction anywhere from the lower end of the sac till nasolacrimal duct
Mucoid regurgitation from either punctum	Obstruction anywhere from the lower end of the sac till nasolacrimal duct with infection

3. A 14-day-old neonate is brought to you with epiphora. She was diagnosed with left-sided congenital nasolacrimal duct obstruction. How will you explain the technique of massage for the same? (3 marks)

- This method is indicated in the first two months of birth when the child presents with epiphora, and this technique is called Crigler massage (Fig. 2.3.2)
- The mother is advised to use her little finger to massage over the lacrimal sac area moving downwards along the ala of the nose
- It is done repetitively as many times as she feeds the child
- This method helps to remove any membranous folds and helps in the re-canalization of the nasolacrimal duct.
- Also, it has a high success rate
- If the child has a mucoid discharge, topical antibiotics should be applied before the technique

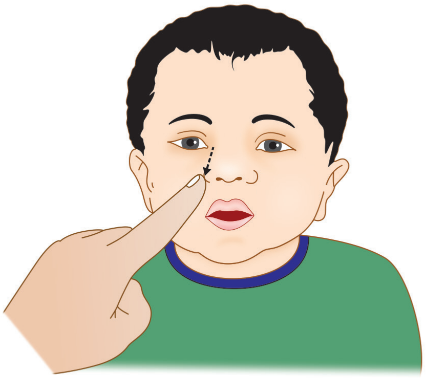


Fig. 2.3.2: Procedure of Crigler massage

COMPETENCY

OP2.4 DESCRIBE THE AETIOLOGY, CLINICAL PRESENTATION. DISCUSS THE COMPLICATIONS AND MANAGEMENT OF ORBITAL CELLULITIS

LONG ESSAY

1. A 40-year-old man presents with gradual onset painful swelling of eyelids associated with mild protrusion of the left eye of one-week duration. He also has fever (temperature 100°F). On examination, there is proptosis, chemosis, and mild restriction of abduction. Anterior segment and fundus are normal.

A. State the most probable diagnosis. (1 mark)
B. Enumerate four causes for the same. (2 marks)
C. Explain how you would like to investigate and manage its patient. (3 marks)
D. Give one differential diagnosis and explain the differences. (4 marks)

A. Most Probable Diagnosis

Left-sided orbital cellulitis

B. Common Causes

1. The spread of infection from the neighboring sinuses is the most common cause
2. Exogenous entry of infection through wound or post-surgical (extraocular surgeries like lid surgeries, dacryocystectomy, or orbitotomy)
3. Spread of infection from teeth
4. Endogenous infection can rarely spread from internal abscesses

C. Investigation and Management

Investigations

- CT scan usually shows an increase in the orbital soft tissue mass, inflammatory stranding of the intraconal fat, edema of the extraocular muscles
- An orbital or periosteal abscess may be seen

Management

- It requires close monitoring to prevent complications. Hospital admission is mandatory. In cases of injury, tetanus toxoid injection has to be administered
- After doing a vitals examination and doing necessary investigations, the patient has to be started on intravenous antibiotics preferably covering gram-positive organisms, like third-generation cephalosporins
- Intravenous steroids are given under the cover of antibiotics (after the first dose of antibiotics) to bring down the orbital inflammation
- Vision and pupil examination have to be done every 4 hours to rule out complications, till the edema comes down
- If there is corneal exposure, tear film substitutes and if required a temporary tarsorrhaphy can be performed. Simple taping of the lids can be very effective
- Surgical intervention should be considered in the following situations:
  - Unresponsiveness to antibiotics
  - Abscess formation
  - Sight threatening disease
  - Atypical presentation may warrant biopsy

D. Differential diagnosis is preseptal cellulitis

Feature	Preseptal cellulitis	Orbital cellulitis
Symptoms	Less pain and swelling, no fever,	More pain, fever
Anterior segment signs	• Lid edema-boggy • Eye movements are normal • No proptosis • Vision is unaffected • Pupil is normal • Color vision is normal • Conjunctiva is congested	• Lid edema-woody • Eye movements are painful and restricted, • Axial proptosis • Vision is affected in later stages • Pupil abnormalities in later stages • Color vision may be affected in later stages • Conjunctiva is chemosed and congested
Posterior segment signs	Normal	Disc edematous
Treatment	Oral antibiotics, analgesics	Oral/intravenous antibiotics Oral steroids under cover of antibiotics
Complications	Less	More

SHORT ESSAY

1. Annotate the clinical features of orbital cellulitis. (5 marks)

Orbital Cellulitis

Inflammation of the contents of the orbit extending beyond the orbital septum

Clinical Features

Symptoms

- Pain, swelling of eyelids are more than in preseptal cellulitis
- When the infection progresses, visual symptoms, protrusion of the eyeball and difficulty in eye movements, inability to lift the upper lid may develop
- There may be other constitutional symptoms like fever and myalgia

Signs

- Lid edema, woody hard
- Conjunctival congestion and chemosis

- Axial proptosis
- Ptosis, ophthalmoplegia, loss of corneal sensation, RAPD, visual diminution, and disc edema are seen in rapidly progressive forms

SHORT ANSWERS

1. Enlist the complications of orbital cellulitis. (3 marks)

Complications of Orbital Cellulitis

Due to spread of infection	Formation of subperiosteal abscess: A collection of pus in the subperiosteal space, the proptosis becomes eccentric Rarely meningeal abscess, cavernous sinus thrombosis
Due to proptosis	Exposure keratopathy
Due to increased intraorbital pressure	Optic nerve compression and central retinal artery/vein occlusion

2. Give the indications of lateral canthotomy in orbital cellulitis and explain the procedure briefly. (3 marks)

Lateral Canthotomy in Orbital Cellulitis

Indications

An increase in orbital pressure due to the formation of an abscess compromises the functioning of the optic nerve as evidenced by a decrease in visual acuity, RAPD, color vision deficit, or a field defect (orbital compartment syndrome).

Procedure

- The patient is made to lie in a supine position after explaining to him the procedure
- Local infiltration anesthesia is injected near the lateral canthus
- An artery forceps is used to crush the tissue at the lateral canthus for around a minute
- This is followed by using scissors to cut the lateral canthus
- The lateral canthal ligament (superior and inferior crux) are also severed (cantholysis) and a decrease in pressure inside the orbit is felt at the eyelids

3. Explain the radiological features of orbital cellulitis. (3 marks)

- The imaging modality of choice in orbital cellulitis is a CT scan of the orbit with paranasal sinus
- The features that can be seen are:
  - An increase in the orbital soft tissue mass
  - Inflammatory stranding of the intraconal fat, edema of the extraocular muscles
  - An orbital or subperiosteal abscess may be seen
  - There may be associated inflammation of the neighboring paranasal sinuses

4. Give the modified Chandler Staging system for orbital inflammation. (3 marks)

Modified Chandler Staging System

Stage I	Preseptal cellulitis
Stage II	Orbital cellulitis
Stage III	Subperiosteal abscess
Stage IV	Orbital abscess
Stage V	Cavernous sinus thrombosis

5. Enumerate the clinical features of orbital apex syndrome. (3 marks)

Orbital Apex Syndrome Consists of the Following Features

- Loss of function of oculomotor nerve: Adduction, elevation, depression along with ptosis and loss of the pupillary reflex
- Loss of function of trochlear nerve: Intorsion
- Loss of function of abducens nerve: Loss of abduction
- Loss of corneal sensations due to affection of ophthalmic branch of the trigeminal nerve
- Diminution of visual acuity/color vision/visual field due to the affection of the optic nerve. Reverse RAPD may be seen in the other eye (absence of consensual light reflex in the other eye)

6. Draw a neat and labeled diagram of orbital apex and structures passing through it. (3 marks)

Orbital Apex

See Figure 2.4.1

7. Enumerate various orbital spaces. If fluid were to accumulate in one of these spaces, there is compromise on the optic nerve function. Which space is this? (2+1 marks)

Orbital Spaces

1. Periorbital space
2. Peripheral space
3. Central or retrobulbar space
4. Sub-Tenon space

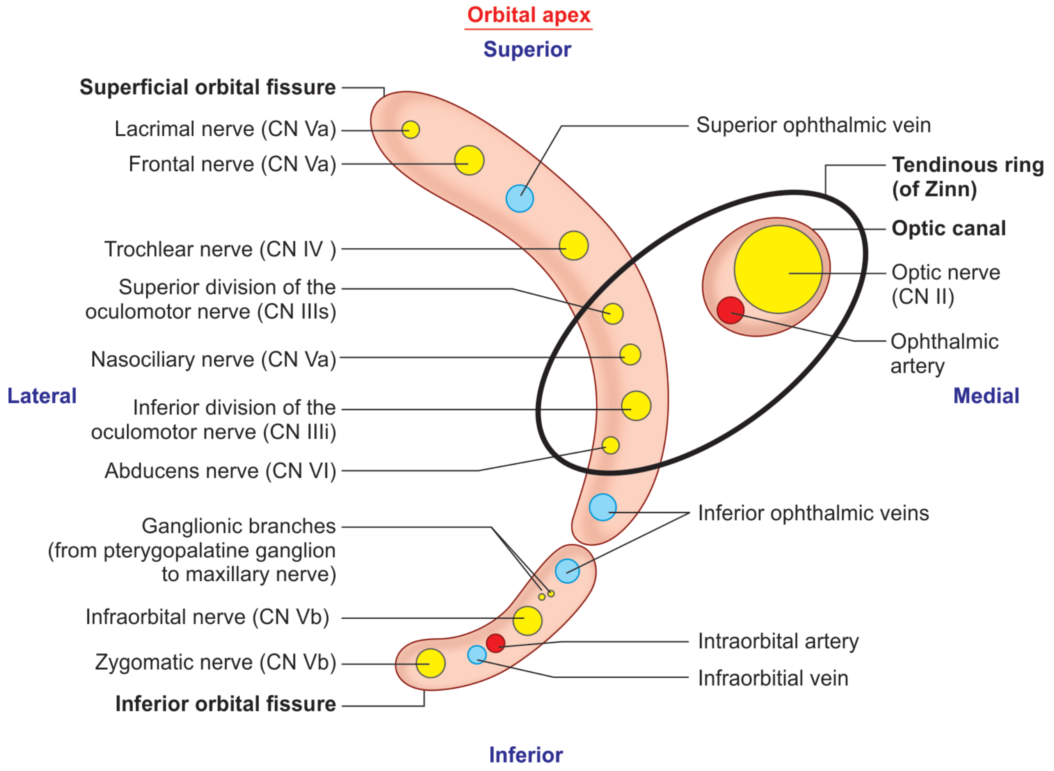

Orbital apex
Superior
Superficial orbital fissure
Lacrimal nerve (CN Va)
Frontal nerve (CN Va)
Superior ophthalmic vein
Tendinous ring (of Zinn)
Optic canal
Trochlear nerve (CN IV)
Superior division of the oculomotor nerve (CN IIIs)
Optic nerve (CN II)
Ophthalmic artery
Nasociliary nerve (CN Va)
Lateral
Medial
Inferior division of the oculomotor nerve (CN IIIi)
Abducens nerve (CN VI)
Inferior ophthalmic veins
Ganglionic branches (from pterygopalatine ganglion to maxillary nerve)
Infraorbital nerve (CN Vb)
Intraorbital artery
Zygomatic nerve (CN Vb)
Infraorbitial vein
Inferior orbital fissure
Inferior

Fig. 2.4.1: Orbital apex

Compromise of the Optic Nerve Function

- The retrobulbar space is a tight compartment around the optic nerve
- If there is a rise in the pressure in this space, the optic nerve function is compromised
- Also, the central retinal artery gets compressed

COMPETENCY

OP2.5 DESCRIBE THE CLINICAL FEATURES ON OCULAR EXAMINATION AND MANAGEMENT OF A PATIENT WITH CAVERNOUS SINUS THROMBOSIS

SHORT ESSAYS

1. Draw a neat-labeled diagram of the cavernous sinus and enumerate its tributaries. (3+2 marks)

Cavernous Sinus (Fig. 2.5.1)

See Figure 2.5.1

Tributaries

1. Anteriorly superior and inferior ophthalmic veins
2. Posteriorly superior and inferior petrosal sinus
3. Transverse sinus connects two cavernous sinuses
4. Superiorly, veins of the cerebrum
5. Inferiorly with pterygoid venous plexus

2. Explain the clinical features of cavernous sinus thrombosis. (5 marks)

Clinical Features of Cavernous Sinus Thrombosis

1. It is usually unilateral but soon becomes bilateral
2. The patient is usually toxic, with features of septicemia
3. Severe pain in the affected eye, with conjunctival congestion, chemosis, and marked proptosis
4. Paralysis of the 6th nerve occurs first as it is present within the substance of the sinus. It is followed by the third nerve, the fourth nerve palsy

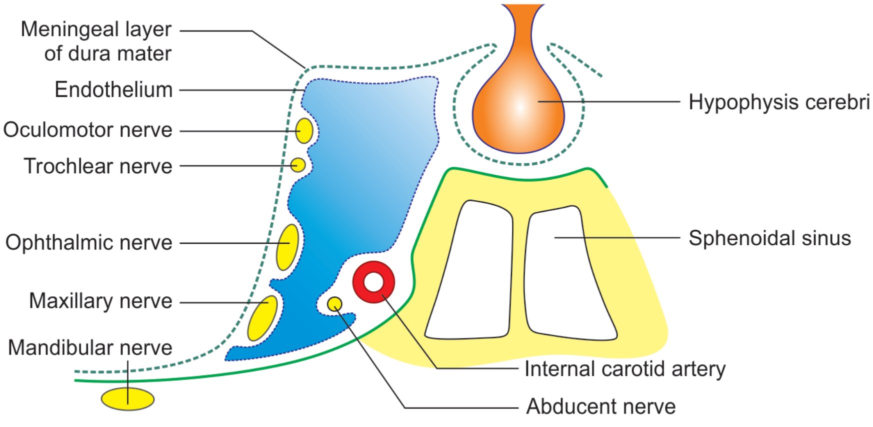

Meningeal layer of dura mater
Endothelium
Oculomotor nerve
Trochlear nerve
Ophthalmic nerve
Maxillary nerve
Mandibular nerve
Hypophysis cerebri
Sphenoidal sinus
Internal carotid artery
Abducent nerve

Fig. 2.5.1: Anatomy and relations of the cavernous sinus

5. There is a loss of sensation in the region supplied by the ophthalmic nerve and maxillary division of the trigeminal nerve. As a result, there is total ophthalmoplegia with loss of sensations over the cornea
6. Edema in the mastoid region due to back pressure
7. Visual loss may occur in later stages due to compression of the chiasm
8. Complications: Meningitis, septicemic shock, death

3. Name one cause of pulsatile proptosis. Explain its clinical features. (1+4 marks)

Pulsatile Proptosis

Caroticocavernous fistula is a cause for pulsatile proptosis

Clinical Features

- High flow fistula usually follows trauma or accidents
- Low flow fistula is spontaneous and seen in the elderly
- There is pulsatile proptosis, chemosis, total ophthalmoplegia with cork-screw vessels over the conjunctiva. Fundus examination shows disc edema and engorged veins
- A thrill is felt and a bruit is heard

SHORT ANSWERS

1. In cavernous sinus thrombosis, abduction is the first document to be affected. Justify. (3 marks)

Or

Give the anatomical basis for affection of the abducens nerve first in a case of cavernous sinus thrombosis (3 marks)

Anatomical Basis (Fig. 2.5.1)

- As seen in the image, the abducens nerve lies within the substance of the cavernous sinus
- As a result, when there is inflammation of the sinus, the abducens nerve is first affected
- As abduction is its main function, abduction is first affected

2. Differentiate orbital apex syndrome from cavernous sinus syndrome? (3 marks)

- Both orbital apex syndrome and cavernous sinus syndrome, there is total ophthalmoplegia (external and internal), with visual loss, and loss of corneal sensation
- The following are three important differences between the two:
  1. The cavernous sinus tends to be bilateral with time
  2. The patient is usually sick with features of septicemia
  3. Most importantly, there is a loss of function of the maxillary division of the trigeminal nerve leading to loss of sensation over the maxillary area

3. Enumerate the radiological features of cavernous sinus thrombosis. (3 marks)

- The best modality to diagnose cavernous sinus thrombosis is an MRI venogram
- On MRI, there is an increase in the size of the cavernous sinus with obvious proptosis on the same side. Also, there is an increase in the diameter of the superior ophthalmic vein
- On an MRI angiogram, there is a filling void seen in the affected cavernous sinus

4. Explain the management of cavernous sinus thrombosis. (3 marks)

- Cavernous sinus thrombosis is an emergency condition and the patient has to be hospitalized. He should be monitored for the development of sepsis through serial blood count and culture if necessary
- Intravenous broad-spectrum antibiotics like vancomycin, third-generation cephalosporin, and metronidazole are given for 3–4 weeks. Even after the features subside, it is recommended to be continued for 2 more weeks
- Analgesics and anti-inflammatory drugs are also given
- Steroids have been tried with minimal success

COMPETENCY

OP2.6 ENUMERATE THE CAUSES AND DESCRIBE THE DIFFERENTIATING FEATURES, AND CLINICAL FEATURES AND MANAGEMENT OF PROPTOSIS

LONG ESSAYS

1. A 45-year-old man presents with bilateral protrusion of the eyeball associated with congestion and chemosis of conjunctiva. He has a staring look on attempting downgaze, the upper right lid lags (lid lag).

A. What is the most probable diagnosis? (1 mark)
B. Explain the pathophysiology of this condition. (3 marks)
C. Explain the WHO staging of this disease. (3 marks)
D. Explain the indications and types of orbital decompression. (3 marks)

A. Most Probable Diagnosis

Thyroid-associated orbitopathy.

B. Pathophysiology (Fig. 2.6.1)

- Thyroid-associated orbitopathy is an autoimmune condition. In this disease, orbital fibroblasts play a key role

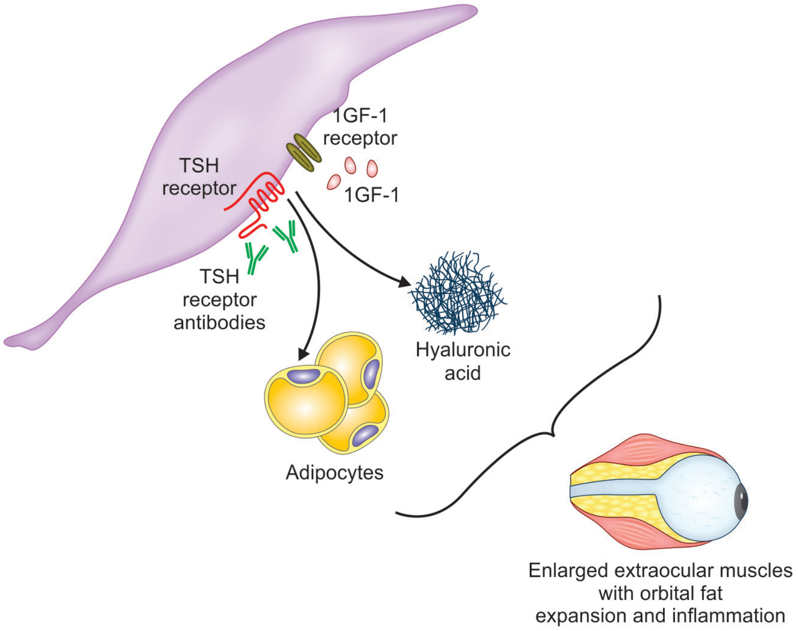

1GF-1 receptor
TSH receptor
1GF-1
TSH receptor antibodies
Hyaluronic acid
Adipocytes
Enlarged extraocular muscles with orbital fat expansion and inflammation

Fig. 2.6.1: Pathogenesis of thyroid orbitopathy

- Autoantibodies are set up against thyroid receptors. As a result, mononuclear cells proliferate
- The reason why the orbit is targeted by these mononuclear cells which have been activated against thyroid follicular cells is not yet known
- One probability is "molecular mimicry" between thyroid follicular cells and orbital fibroblasts
- A reaction between the activated mononuclear and local fibroblasts results in the release of cytokines like TNF-α, IFN-γ, and IL-1β into the surrounding
- They stimulate the expression of immunomodulatory proteins in orbital fibroblasts, thus perpetuating the autoimmune response in the orbital connective tissue
- As a result, there is an increase in connective tissue volume, increased deposition of glycosaminoglycans, and the fibrotic restriction of extraocular-muscle movements

C. WHO Staging of Thyroid-associated Orbitopathy

Class 0	No signs and symptoms
Class I	Only signs, no symptoms (mainly lid related signs)
Class II	Soft tissue involvement and symptoms like lacrimation, photophobia
Class III	Proptosis
Class IV	Involvement of extraocular muscles
Class V	Corneal involvement (exposure)
Class VI	Sight loss due to optic nerve involvement

Mnemonic: NO SPECS

D. Indications and Types of Orbital Decompression

Indications

1. Severe progressive proptosis causing optic nerve damage to the optic nerve or exposure keratopathy
2. Presence of thyroid-associated optic neuropathy (visual loss, RAPD, or visual field defects) due to the enlarged muscles compressing the optic nerve

Types

1. Two-wall: Antral-ethmoidal: removal of part of the floor and posterior part of the medial wall, provided around 5 mm of decrease in proptosis
2. Three-wall: Two-wall + lateral wall: decreases around 6–10 mm
3. Four-wall: Three-wall + lateral half of roof+ large part of sphenoid at the apex: decreases 12–16 mm proptosis

2. Enumerate the differential diagnosis for proptosis. Describe the work-up of a case of proptosis. (4+6 marks)

Differential Diagnosis for Proptosis

Unilateral	Bilateral
Congenital • Dermoid cyst, teratoma ***Traumatic*** • Orbital hemorrhage, traumatic aneurysm, orbital emphysema	***Systemic*** • Thyrotoxicosis, histiocytosis, systemic amyloidosis, xanthomatosis, and Wegener's granulomatosis

Contd.

Unilateral	Bilateral
Inflammatory • Orbital cellulitis, abscess, subperiosteal abscess, panophthalmitis, and cavernous sinus thrombosis • Pseudotumors, tuberculoma, gumma, and sarcoidosis ***Vascular*** • Angioneurotic edema, orbital varix, and aneurysms ***Neoplasms*** • Lacrimal tumors, lymphomas, glioma, meningioma, neurofibroma, rhabdomyosarcoma, spread from tumors from adjacent tumors, metastases ***Cysts*** • Implantation cyst and parasitic cysts, mucoceles	***Inflammatory*** • Established stages of cavernous sinus thrombosis ***Neoplasms*** • Bilateral metastases from neuroblastoma, nephroblastoma, Ewing's sarcoma, and leukemic infiltration ***Others*** • Craniofacial dysostosis , rickets, osteitis ***Pulsating proptosis*** • Carotico-cavernous fistula • Meningocele • Encephalocele ***Acute proptosis*** • Orbital emphysema • Orbital hemorrhage • Sudden rupture of mucocele ***Intermittnt proptosis*** • Orbital varix • Periodic orbital edema • Recurrent orbital hemorrhage and highly vascular tumors

Work-up of a Case of Proptosis

A. History Taking

- A detailed history regarding the mode of onset of proptosis, whether associated with trauma, fever, and pain must be taken
- History regarding other systemic disorders should be asked for
- History regarding visual disturbances, diplopia, and transient visual loss must be asked

B. Examination

- A complete ophthalmological examination should be carried out
- Some special instances concerning examination have been exemplified below

Clinical featues	Site of pathology/disease
Visual impairment, RAPD, color vision disturbances	Lesions at the optic nerve
Restricted extraocular motility	Intraconal or extraconal lesions
Axial proptosis Hypermetropia, macular edema, choroidal folds	Intraconal lesions
Eccentric proptosis	Extraconal lesions
Fundus: disc edema, optico-ciliary shunts	Chronic compression around the optic nerve (meningioma)
Central retinal artery pulsating	Acute severe compression around the optic nerve
Loss of corneal sensation	Consider superior orbital fissure or orbital apex lesions
Presence of corkscrew vessels over the conjunctiva	Caroticocavernous fistula

C. Examining Proptosis

1. **Inspection:** Rule out pseudoproptosis, ascertain whether the proptosis is unilateral or bilateral; note any abnormality in the shape of the skull; and observe whether proptosis is axial or eccentric
2. **Palpation:** To know compressibility of the tumor, for an orbital thrill, for any swelling around the eyeball, palpate regional lymph nodes and orbital rim to note any thickening or loss of continuity
3. **Auscultation:** It is primarily of value in searching for abnormal vascular communications that generate a bruit, such as carotico-cavernous fistula
4. **Transillumination:** It helps evaluate anterior orbital lesions
5. **Special tests:** Valsalva maneuver—to evaluate the size of tumors in relation to Valsalva

D. Measurement of Proptosis

- It is called exophthalmometry. It is done to know the severity of proptosis and to know if proptosis is axial or eccentric
- Clinically, proptosis is assessed by standing behind the patient and tilting his head slightly behind to observe both eyes
- The instruments used for measurement of proptosis are
  - *Hertel's exophthalmometry:* This is mainly used to measure eccentricity (8:37)
  - *Luedde's exophthalmometry:* The apex is placed on the lateral orbital margin and measurement is taken at a point where the corneal apex coincides

E. Investigations

1. **Blood investigations:** Include complete blood count, thyroid hormone levels, thyroid antibodies, serum calcium, VDRL test, stool test to rule out ova and cysts, urine tests to rule out Bence Jones proteins
2. **Radiological investigations**

Type of investigation	Utility
X-ray of the orbit	Orbital cavity, enlargement of optic foramina, calcification, and hyperostosis. Frontal view—to compare two orbits Caldwell view—to visualize superior orbital fissure Water's view—to study orbital floor fractures Rheese view—to visualize optic foramen Lateral view—nasopharynx
Computed tomography of orbit	Most commonly used, more information than X-ray. Gives the exact size and location of the lesion. Also to look at paranasal sinuses, nasopharynx, anterior cranial fossa
Magnetic resonance imaging	To visualize orbital apex, optic nerve
Ultrasonography or echography	To visualize solid, cysts, infiltrative masses
MR venogram	Helpful in orbital varix
Carotid angiogram	Caroticocavernous fistula

3. **Tissue diagnosis:** For definitive diagnosis

Fine-needle aspiration biopsy (FNAB) guided by CT/ultrasound	Histopathological diagnosis in orbital tumors
Incisional biopsy	At least 5–10 mm of tissue required, infiltrative lesions
Excision biopsy	The preferred technique for well-encapsulated tumors

SHORT ESSAYS

1. Explain methods of measurement of proptosis. (5 marks)

- Proptosis is a forward displacement of the eyeball beyond the orbital margins
- Normal value: 21–25 mm. A discrepancy of 2 mm between the two eyes is needed to define proptosis
- Measurement of proptosis is called exophthalmometry and is done to know the severity of proptosis and to know if proptosis is axial or eccentric
- Clinically, proptosis is assessed by standing behind the patient and tilting his head slightly behind to observe both eyes
- The instruments used for measurement of proptosis are:
    - *Hertel's exophthalmometry:* This is mainly used to measure eccentricity
    - *Luedde's exophthalmometry:* The apex is placed on the lateral orbital margin and measurement is taken at a point where the corneal apex coincides
- While measuring proptosis, the following measurements are done:
    a. AP measurement (from the lateral orbital margin to the apex of the cornea) in mm
    b. The measurement from the highest point of the superior orbital margin to the center of the pupil (to measure vertical dystopia)
    c. Measurement from the center of the nasal bridge to the center of the pupil (to measure horizontal dystopia)

Eye	Measurement (a)	Measurement (b)	Measurement (c)
Right eye			
Left eye			

- This is a simple way of charting measurements and helps in deciding whether the proptosis is axial or eccentric, and if eccentric, whether vertical or horizontal
- Difference in column a, between the right and the left eye determine if
- If the measurements in the second column (b) differ, the eccentricity is vertical, whereas if a discrepancy is seen in the third column (c), eccentricity is horizontal

2. Explain the clinical features and radiological features of orbital pseudotumor. (5 marks)

- Orbital pseudotumor is an old terminology for Nonspecific idiopathic orbital inflammatory disorder (NSOIDs) and includes a group of non-neoplastic, non-infectious orbital space-occupying inflammatory lesions
- They are characteristically self-limiting, recurrent, non-neoplastic, rapidly progressing, and respond well to systemic steroids
- It is a diagnosis of exclusion, i.e., other causes for proptosis should be carefully excluded before labeling a patient with NSOID

Clinical Features

- It usually affects individuals in their fourth decade, may be unilateral or bilateral. In pediatric cases, it is usually bilateral and may be associated with eosinophilia
- Patients present with abrupt onset of pain (most common), periocular redness, proptosis, chemosis, and lid edema

- Signs depend on the location of the orbit that is affected
  - The affection of extraocular muscles (myositis) present with painful restriction of one or more extraocular movements, with proptosis. Medial rectus is the most commonly involved muscle. Vascular injection over the involved muscle is characteristic of this subtype
  - The affection of orbital apex causes orbital apex syndrome with minimal proptosis and total ophthalmoplegia, pupillary dilatation, and loss of vision. On examination, there is loss of corneal reflex, disc edema, and choroidal folds additional to the common clincial features
  - The affection of lacrimal gland (acute dacryoadenitis) presents with painful eccentric proptosis (eye is down and out), tenderness in the superotemporal aspect of the orbit, and vascular injection in the upper palpebral conjunctiva close to the lacrimal gland.
- These features are usually self-limiting and there is usually a history of recurrence.
- Severe and multiple attacks of inflammation can lead to fibrosis of tissues leading to a contracted globe with loss of motility and is referred to as 'Frozen Orbit'

Radiological Features

Orbital myositis	MRI/CT scans show diffuse enlargement of the involved muscle in contrast to thyroid eye disease, where there is sparing of the tendinous attachments
Orbital apex	MRI/CT scans show inflammatory infiltrate of the orbital apex
Dacryoadenitis	MRI/CT scans show enlargement of the lacrimal gland without bony erosion

3. Enumerate the eye signs of thyroid ophthalmopathy. (5 marks)

1. **Lid signs**
   i. Dalrymple sign: Staring look due to lid retraction
   ii. von Graefe sign: Lid lag during downgaze
   iii. Enroth's sign: The fullness of lids
   iv. Gifford's sign: Inability to evert the upper lid
   v. Stellwag sign: Infrequent blinking
   vi. Jellinek's sign: Increased pigmentation of the lids
   vii. Joffroy's sign: Poor forehead wrinkling on upgaze
2. **Conjunctival signs**
   - Congestion
   - Chemosis
3. **Pupil**
   - RAPD may be present in presence of neuropathy
4. **Ocular motility**
   - There is restrictive strabismus, i.e., incomitant squint due to restriction. The inferior rectus is the most common muscle involved and the first sign to occur is a deficit of uniocular elevation
   - Mobius's sign is an inability to converge eyes
5. **Proptosis**
   - Can be unilateral, or bilateral, symmetric, or asymmetric, and is usually axial
6. **Optic neuropathy and exposure keratopathy**
   - Complications

4. Describe the EUGOGO classification of thyroid ophthalmopathy. (5 marks)

EUGOGO is a European group of Graves' orbitopathy classification and is based on the quality of the patient's life and the risk of vision loss

Mild TAO Minimum impact on patient's life	• Minor lid retraction • Mild soft-tissue involvement • <3 mm exophthalmos • No diplopia • Corneal exposure responsive to lubricants
Moderate TAO Have an impact on patient's life, but the disease is not sight-threatening yet	• Lid retraction >2 mm • Moderate to severe soft-tissue involvement • Exophthalmos >3 mm • Inconstant diplopia
Severe or sight-threatening TAO	• Optic neuropathy • Corneal breakdown

5. Explain the surgical management of Graves' ophthalmopathy. (5 marks)

Surgical intervention for thyroid ophthalmopathy is indicated in two circumstances:

1. In the active phase, when there is compression of the optic nerve or there is exposure keratopathy
2. In the quiescent phase, for correction of restrictive strabismus

Active phase	**Quiescent phase**
Orbital decompression • To decrease the orbital pressure • Types 1. *Two-wall:* Antral-ethmoidal: removal of part of the floor and posterior part of the medial wall, provided around 5 mm of decrease in proptosis 2. *Three-wall:* Two-wall + lateral wall: decreases around 6–10 mm 3. *Four-wall:* Three-wall + lateral half of roof+ large part of sphenoid at the apex: decreases 12–16 mm proptosis	***Squint surgery*** • Indicated after orbital decompression • A recession of the affected muscle should be performed ***Eyelid surgery*** • Botulinum toxin injection can be done for temporary relief from lid retraction • After squint surgery, eyelid surgeries should be performed • Mullerectomy: Disinsertion of Muller's muscle in lid retraction • Levator disinsertion: Moderate to severe lid retraction • The recession of lower lid retractors for low lid retraction • Blepharoplasty for removal of excess fatty tissue and redundant skin around the eyelid

SHORT ANSWERS

1. Describe the clinical features of the Tolosa–Hunt syndrome. (3 marks)

- Tolosa–Hunt syndrome is a rare, idiopathic granulomatous inflammation of the orbital apex
- Patients present with recurrent episodes of orbital pain with ophthalmoplegia with minimal or no proptosis
- Pupillary involvement is also present. All the signs of orbital apex syndrome are present

- Diagnosis is established by MRI or histopathology of the lesion at the orbital apex
- MRI shows granulomatous inflammation of the orbital apex and an increase in the size of the cavernous sinus
- Clinical response is prompt with systemic steroids and is seen within 48 hours and evidenced on MRI

2. Enumerate three causes of pseudoproptosis. (3 marks)

1. Axial myopia
2. Contralateral enophthalmos
3. Contralateral ptosis

3. Explain how to differentiate between axial and eccentric proptosis. (3 marks)

- To differentiate between axial and eccentric proptosis, the following chart is used

Eye	AP measurement (from the lateral orbital margin to the apex of the cornea) in mm	The measurement from the highest point of the superior orbital margin to the center of the pupil (vertical dystopia)	Measurement from the center of the nasal bridge to the center of the pupil (horizontal dystopia)
Right eye			
Left eye			

- This is a simple way of charting measurements and helps in deciding whether the proptosis is axial or eccentric, and if eccentric, whether vertical or horizontal
- In axial proptosis, the measurements in the second and the third column are equal in both the right eye and left eye
- If the measurements in the second column differ, the eccentricity is vertical, whereas if a discrepancy is seen in the third column, eccentricity is horizontal

4. Describe briefly, the clinical features of a craniofacial anomaly that can present with pseudoproptosis. (3 marks)

- Crouzon syndrome is an autosomal dominant condition and is the most common craniofacial dysostosis
- It is characterized by a short AP diameter of the skull (brachycephaly), shallow orbits, maxillary hypoplasia
- There is pseudoproptosis due to shallow orbit, hypertelorism, and V pattern exotropia
- Optic atrophy may ensue due to compression of the optic nerve at the optic canal
- Other features include ectopia lentis, cataracts, buphthalmos, developmental delay, mental retardation, high arched palate, and irregular dentition

5. Enumerate three causes for enophthalmos. (3 marks)

1. Blow-out fracture
2. Cicatrizing variant of orbital pseudotumor
3. Horner's syndrome

COMPETENCY

OP2.7 CLASSIFY THE VARIOUS TYPES OF ORBITAL TUMOURS. DIFFERENTIATE THE SYMPTOMS AND SIGNS OF THE PRESENTATION OF VARIOUS TYPES OF OCULAR TUMOURS

SHORT ANSWERS

1. Describe the clinical features and management of orbital dermoid. (3 marks)

Orbital Dermoid

Dermoid is a common developmental benign tumor that arises because of displacement of the epidermis into a subcutaneous plane.

Clinical Features

They usually present in the first or second decade of life with no gender predilection

Symptoms

- A painless mass in the superolateral or superomedial aspect of the orbit
- It is usually non-progressive

Signs

- On examination, the mass is usually in the superolateral or superomedial quadrant of the orbit
- It is well-circumscribed, free from underlying bone, and the skin is pinchable over the swelling
- There are two clinical variants
    - Superficial
    - Deep
- The superficial variant is anterior to the orbital septum and does not cause dystopia. Also, there are no associated bony defects
- The deep dermoid, on the other hand, present with proptosis and thus the posterior margins cannot be fully traced clinically

Management

- A CT scan helps to differentiate the two clinical variants through clinical examination suffices
- The treatment of choice is total excision of the dermoid by lateral or medial orbitotomy
- Care must be taken to remove the entire contents of the dermoid

2. Describe the clinical features of optic nerve glioma. (3 marks)

Optic Nerve Glioma

It is a benign tumor arising from the astrocytes, very commonly associated with neurofibromatosis.

Clinical Features

Symptoms

- It usually occurs in the first decade of life
- Around a quarter of the patients have Neurofibromatosis I
- Children present with a visual loss followed by proptosis

Signs

- The proptosis is usually axial and there is usually an RAPD
- Fundus examination shows primary optic atrophy due to the tumor compressing the ipsilateral optic nerve
- The tumor has a propensity to spread posteriorly towards the chiasm, hypothalamus, and midbrain
- Thus, a careful follow-up in the form of visual field testing is recommended

3. Describe the clinical features and management of rhabdomyosarcoma. (3 marks)

Rhabdomyosarcoma

- It is the most common orbital tumor in childhood
- It is a highly malignant tumor arising from the pluripotent mesenchymal elements of the eye

Clinical Features

Symptoms

It presents as rapidly progressive proptosis of sudden onset in a child of 7–8 years

Signs

- The tumor commonly involves the superonasal quadrant but may arise from any part of the orbit
- It mimics inflammation as the skin over the swelling is warm
- However, there is no rise in temperature that helps to differentiate from inflammatory conditions

Management

- The clinical suspicion is supported by X-rays showing bone destruction and CT scan demonstrating tumor concerning an extraocular muscle
- Diagnosis is confirmed by biopsy
- If the tumor is well-circumscribed, then complete resection is possible. Combined radiation and chemotherapy is provided in severe cases
- Chemotherapy has a good outcome in the embryonal variant on histopathology
- Exenteration is reserved for unresponsive cases

4. Describe the clinical features of orbital lymphoma. (3 marks)

Orbital Lymphoma

- Lymphomas are malignant tumors of lymphoreticular origin and have a plethora of clinical presentations
- Orbits are involved more commonly by non-Hodgkin's lymphomas
- It may involve lacrimal glands, lids, or subconjunctival tissue

Clinical Features

Symptoms

- It usually presents in the 5th or 6th decade with slow progressive unilateral/bilateral proptosis
- There are no other symptoms

Signs

- Proptosis may be axial or eccentric, depends on the site of affection of the orbit
- A salmon-colored tissue is usually seen in the subconjunctival space if the anterior orbit is involved. The tumor is rubbery in consistency
- If the posterior orbit is involved, there is proptosis and restricted ocular motility. These tumors 'hug the eyeball', i.e., they indent the eyeball, and usually, no invasion is seen

5. Enumerate three benign orbital tumors. (3 marks)

1. Dermoid
2. Pleomorphic adenoma of the lacrimal gland
3. Cavernous hemangioma

6. Enumerate three common causes of orbital metastasis. (3 marks)

1. Neuroblastoma
2. Nephroblastoma
3. Ewing's sarcoma

COMPETENCY

OP2.8 LIST THE INVESTIGATIONS HELPFUL IN DIAGNOSIS OF ORBITAL TUMORS. ENUMERATE THE INDICATIONS FOR APPROPRIATE REFERRAL

SHORT ESSAYS

1. Describe the utility of CT scans and MRI in orbital tumors with examples. (5 marks)

Utility of CT Scan

- CT scan holds prime importance in investigating orbital disorders, ranging from inflammatory to malignant conditions. CT scan gives the exact location of the tumor
- CT scan provides axial and coronal views of various cross-sections of the orbit
- An axial section is a horizontal plane that is perpendicular to the cephalocaudal axis. Axial views are taken through the inferior most of orbit, the lower part of the orbit, mid-orbit, superior orbit, and roof of the orbit
- The coronal section is taken parallel to the cephalocaudal axis. It is done anterior-most of the orbit, anterior orbit, central orbit, anterior orbital apex, and posterior orbital apex

Utility of MRI

- MRI of the orbit is indicated to study the soft tissue masses, orbital apex, and optic nerve pathologies
- In addition to axial and coronal sections, MRI provides a view in the sagittal section too
- These investogations help to clinch the diagnosis when aided with a good clinical work-up

Tumor	CT/MRI
Cavernous hemangioma	Well circumscribed within the muscle cone
Lymphangioma	Well encapsulated cystic tumors, with internal septations and fluid-fluid levels
Optic glioma	Fusiform enlarged tumor, kinking of the optic nerve, may extend till chiasm and beyond
Neurofibroma	Hyperintense usually extraconal mass

Contd.

Tumor	CT/MRI
Meningioma	Tubular enlargement of the optic nerve with calcification: Tram track appearance If sphenoid wing meningioma, dural extension into the chiasm can be seen
Rhabdomyosarcoma	The tumor is isodense with the extraocular muscle eroding into neighboring structures
Lymphoma	Homogenous enhancing mass does not invade bone or neighboring structures, indents the globe
Pleomorphic adenoma	Well encapsulated rounded mass in the lacrimal gland may erode bone in long-standing cases

2. Describe the utility of tissue diagnosis in the evaluation of orbital tumors with examples. (5 marks)

- In orbital tumors, definitive diagnosis is established after histopathological diagnosis
- It can be obtained from an incisional biopsy or after the excision of the tumor
- Histopathology also helps to decide the type of treatment for the particular tumor
  - For example, in a rhabdomyosarcoma, the embryonal variant is sensitive to chemotherapy
  - Histopathology along with cytological markers helps in the diagnosis of the subtypes of tumors and thus helps in prognosticating the tumor

Tumor	Characteristics on Histopathology
Cavernous hemangioma	A well-encapsulated tumor composed of large cavernous spaces containing RBC, walls of spaces is made up of smooth muscle
Lymphangioma	Large serous fluid-filled channels lined by flat endothelial cells
Optic glioma	Astrocytic proliferation, arachnoid hyperplasia
Neurofibroma	spindle-shaped cells arranged in ribbons and cords among a background of the loose myxoid stroma
Meningioma	Syncytial growth pattern, composed of cells with indistinct cytoplasmic margins and Psammoma bodies (round calcifications surrounded by meningioma cells) may be present
Rhabdomyosarcoma	Undifferentiated pluripotent mesenchymal elements can have spindle cells or fibrovascular strands which stacked rhabdomyoblasts or pleomorphic cells
Lymphoma	Excision biopsy is preferred, lymphoid hyperplasia and abnormal clonal expansion of precursor cells
Pleomorphic adenoma	Mixed variety of cell morphology, the proliferation of benign epithelial cells in a stroma composed of cartilaginous, mucinous, or osteoid degeneration

SHORT ANSWERS

1. Describe radiological findings of

A. Optic nerve sheath meningioma **(1 mark)**

B. Optic nerve glioma **(1 mark)**

C. Orbital lymphoma **(1 mark)**

A. Optic nerve sheath meningioma	Tubular enlargement of the optic nerve with calcification: tram track appearance If sphenoid wing meningioma, dural extension into the chiasm can be seen

Contd.

B. Optic nerve glioma	Fusiform enlarged tumor, kinking of the optic nerve, may extend till chiasm and beyond
C. Orbital lymphoma	Homogeneous enhancing mass does not invade bone or neighboring structures, indents the globe

2. Describe the characteristic histopathology findings in different types of rhabdomyosarcoma. (3 marks)

Rhabdomyosarcoma has four types of histopathological variants:

1. **Embryonal:** Striated muscle differentiation, the most common variant, has a good prognosis
2. **Alveolar:** Lesser number of cells show skeletal muscle differentiation, the prognosis is worse than embryonal
3. **Botryoid:** More common in infants, 'grape-like clusters' of cells
4. **Pleomorphic:** Admixtures of many types of cells

SHORT ESSAYS (MISCELLANEOUS)

1. Enumerate three common malignant lid tumors. Describe the clinical features of each of them. (1+4 marks)

Common Malignant Lid Tumors

1. Basal cell carcinoma
2. Squamous cell carcinoma
3. Sebaceous cell carcinoma

Basal cell carcinoma	Squamous cell carcinoma	Sebaceous cell carcinoma
• Most common malignant tumor of the lids (90%) in elderly people • It is locally invasive and does not metastasize • They involve the lower lid (50%), medial canthus (25%), upper lid (10–15%), and outer canthus (5–10%) • They usually present as a noduloulcerative mass with pearly rolled margins • It burrows deep and may invade underlying bones • Other rare presentations include non-ulcerated nodular form, sclerosing or morphea type, and pigmented basal cell carcinoma	• It is second to BCC in its incidence • Risk factors include sun exposure, xeroderma pigmentosum, and dysplastic conditions • It commonly arises from the lid margin (mucocutaneous junction) in elderly patients • Affects upper and lower lids equally • They may present with an ulcerative growth with elevated and indurated margins or as a fungating or polypoid verrucous lesion without ulceration • They metastasize to regional lymph nodes	• It is also a common tumor of the lid • They present as chronic blepharitis • Usually, it is unilateral and causes severe distortion of the lid margin (as against blepharitis) • It has a pagetoid spread, that is the infiltration of the skin, and thus can be diffusely involved

2. Describe the etiopathogenesis, clinical features, and management of orbital mucormycosis. (1+2+2 marks)

Orbital Mucormycosis

It is a life-threatening, sight-threatening fungal infection of the orbit usually seen in debilitated patients, post-renal transplant (on steroid/immunosuppressant), uncontrolled diabetes

Etiopathogenesis

- The causative organisms are *mucor* and *Rhizopus*
- The infection spreads due to fungal spores entering the nasal cavity and invading the sinuses
- They enter the orbit through the inferior orbital fissure or by direct destruction of bone and soft tissue
- After eroding all the structures in orbit, the infection spreads to the intracranial cavity either through the orbital apex or by direct erosion of bone

Clinical Features

- It is usually seen in immunocompromised individuals, without any age or sex predilection
- Patients on long-term systemic steroids and immunosuppressives are at high risk
- It is usually unilateral, however, may be bilateral in 15% of cases
- Initial symptoms are lid edema, painful restriction of one or two eye movements, with mild chemosis
- Pain and proptosis increase, with more and more restriction of eye movements
- Central retinal artery occlusion causes loss of vision
- Meanwhile, necrotic areas with black eschar may be seen on the mucosa of the palate, turbinates and nasal septum, and skin of eyelids
- Soon, a picture of orbital apex syndrome develops (total ophthalmoplegia, visual loss, loss of sensation, complete ptosis), chemosis, and necrotic tissue over the eyelids
- If not treated energetically, the patient develops cerebritis, meningitis, brain abscess, and succumbs within days to weeks

Management

- Early stages can be treated with periocular amphotericin B with intravenous amphotericin B
- In later stages when the orbital apex is involved, it requires exenteration
- The prognosis is grave (15–20% mortality)
- Other antifungals that can be used are Posaconazole and Isavuconazole

3. Describe clinical features of five pathologies due to trauma to the orbit. (5 marks)

Pathology and Clinical Features

Pathology	Clinical features
Orbital emphysema	• It is a benign condition in which forceful entry of air into the orbital cavity • It is usually from surrounding air sinuses following fracture of walls of the orbit • The air gets trapped and forms a ball-valve mechanism • This can lead to eccentric proptosis, visual deterioration, and ophthalmic artery occlusion • The severity depends on the amount of air entrapped
Orbital hemorrhage/retrobulbar Hemorrhage	• It may follow orbital fractures or following retrobulbar injections • The symptoms and signs depend on the amount of bleed and its position • They present with severe congestion and chemosis of conjunctiva, ophthalmoplegia, and proptosis

Contd.

Pathology	Clinical features
Traumatic optic neuropathy	• It can occur due to penetrating or blunt trauma • It is characterized by deceased visual acuity, dyschromatopsia, and RAPD • Fundus finding depend on the site of injury to the optic nerve • Optic disc can be normal appearing if the injury is distal to the entry of the central retinal artery • Fundus finding can mimic CRAO if the site of injury is distal to the entry of the central retinal artery
Orbital compartment syndrome	• It is due to an increase in contents in the retrobulbar space • Since it is a small space, an increase in pressure becomes detrimental to the optic nerve perfusion • Symptoms are headache, visual deterioration • Signs are RAPD, field defects, raised intraocular pressure, pulsating retinal vessels, and obstruction • If not corrected, vision can be permanently lost due to CRAO or optic atrophy
Orbital foreign bodies	• Foreign bodies include gun pellets, and fragments of pencil, pen, wood, nail, and glass • They usually present with a chronic discharging sinus and signs of orbital inflammation of varying degrees • Other symptoms are pain, impairment of vision, limitation of movement, proptosis • The presence of a foreign body can be confirmed using radiological methods like X-ray, CT scan, and MRI • As such intraorbital foreign bodies carry the risk of infection

4. Explain the etiology, clinical features, and management of blow-out fracture. (1+2+2 marks)

Blow-out Fracture

Etiology

- When the orbital margins are intact, but the walls are compromised
- Usually results from blunt trauma with an object of size bigger than the base of the orbit
- The force of the blow is transmitted into the orbit leading to the backward displacement of the globe and an increase in intraorbital pressure, leading to fracture of the weakest point of the orbital wall
- The most involved walls are medial (owing to thinness) and floor (owing to a deficiency-infra orbital fissure)
- The contents may prolapse into the neighboring sinus, and the muscle belly may be entrapped by a fractured bony segment

Clinical Features

Symptoms

There is a history of trauma to the eye and patients present with pain, eye swelling, and sometimes diplopia.

Signs

- Signs of trauma: Periorbital edema and ecchymosis, subcutaneous emphysema

- Signs of blow-out fracture depend on the wall that is involved
    - Restricted eye movement in the opposite gaze as the muscle (e.g., restricted upgaze in floor fracture) and associated diplopia may be present in primary gaze or secondary gaze
    - Enophthalmos due to a decrease in orbital contents
    - Paraesthesia of the area supplied by the infraorbital nerve can be seen in floor fractures

Management

Investigations

- Forced duction test
- X-ray orbit water's view may show the "tear-drop" sign- due to prolapse of orbital contents into the maxillary antrum
- CT scan and MRI helpful in locating the exact site of fracture and impingement of muscle, if any

Treatment

- Anti-inflammatory drugs to decrease the pain and inflammation
- Usually observed for 5–10 days for the edema to reduce
- Oral steroids are given to decrease inflammation and limit fibrosis in presence of muscle impingement
- Surgery involves the repair of the floor with allogenic or autogenous materials

Indications of emergency repair of fracture

1. Impinged muscle belly triggering an oculocardiac reflex, nausea, and vomiting
2. Severe vertical diplopia in primary gaze

Indications for Elective Repair

1. Inability to elevate the globe, diplopia within 30 degrees of primary gaze
2. Significant enophthalmos (>2 mm)

SHORT ANSWERS

1. Enumerate the types of orbital fractures. (3 marks)

Types of Orbital Fractures

1. **Orbital wall fracture**
    i. Roof fracture
    ii. Medial wall fracture
    iii. Zygomatic fracture
    iv. Le Fort fractures—types I, II, III
2. **Blow-out fracture**
    - Can involve any wall of the orbit
    - Most commonly medial wall and floor

2. Describe the clinical features of blepharophimosis syndrome. (3 marks)

Blepharophimosis Syndrome: Clinical Features

- Blepharophimosis syndrome is an autosomal dominant condition characterized by congenital anomalies of the eyelid
- It is also known as the BPES syndrome (Blepharophimosis, Ptosis, Epicanthus Inversus Syndrome)

- The main features are
  1. Blepharophimosis or horizontal shortening of the palpebral fissure
  2. Severe bilateral ptosis, that may be symmetrical or asymmetrical
  3. Epicanthus inversus (a fold of skin medial aspect of the lower lid ascending upwards)
  4. Telecanthus (increased distance between the medial canthi of both eyes)
- Other features may include ectropion, high arched eyebrows, strabismus, and lop ears

3. Define the following:

A. Distichiasis **(1 mark)**

B. Epicanthus **(1 mark)**

C. Cryptophthalmos **(1 mark)**

A. Distichiasis

An abnormal extra row of lashes posterior to the existing lash line

B. Epicanthus

A semicircular fold of skin that covers the upper portion of the medial canthus

C. Cryptophthalmos

Failure of the eyelids to form and skin passes continuously over the globe hiding the globe

4. Describe the clinical features of capillary hemangioma of the lid. (3 marks)

Capillary Hemangioma of the Lid

- Capillary hemangioma of the lid is the most common benign tumor
- It occurs at or shortly after birth, often grows rapidly, and in many cases resolves spontaneously by the age of 7 years
- They may be superficial or deep
- The superficial ones are bright red, and the deep ones appear bluish
- They appear as round raised lesions that can be reduced by applying pressure and swell when the child cries
- They may be associated with visceral hemangiomas
- If present on the upper lid, can cause ptosis and lead to amblyopia

5. Describe the clinical features and management of orbital compartment syndrome. (2+1=3 Marks)

Orbital Compartment Syndrome

- Orbital compartment syndrome is an ophthalmic emergency and is caused due to increase in contents in the retrobulbar space
- Since the retrobulbar space is a small space, any increase in pressure becomes detrimental to the optic nerve perfusion
- Common causes are orbital hemorrhage, emphysema, foreign bodies, and sudden expansion of a tumor

Clinical Features

- Symptoms are headache, diplopia, and visual diminution
- The eye is proptosed

- There is chemosis, conjunctival congestion, and hemorrhage (if associated with trauma)
- The cornea may appear dry due to exposure keratopathy
- Extraocular movements are restricted
- The pupil is dilated and non-reactive
- Fundus examination reveals cherry red spot if associated with a central retinal artery occlusion (due to raised orbital pressure)

Management

- Lateral canthotomy and cantholysis to decrease the pressure
- Division of the orbital septum to relieve further pressure
- Excavation of the bleed/air/foreign body that is causing the compartment syndrome

6. Explain the management of NSOIDs or orbital pseudotumor. (3 marks)

Orbital Pseudotumor

NSOIDs include a group of non-infectious, non-neoplastic inflammatory disorders of the orbit characterized by proptosis, pain, and ophthalmoplegia.

Management

- Observation: Mild disease, and if remission is expected
- Nonsteroidal anti-inflammatory drugs, before starting steroids
- Systemic steroids: They should be started only after exclusion of other disorders, as steroids can mask signs of other inflammation and promote infectious etiology
- In case of no remission due to steroids, a low dose of radiotherapy may be administered. It is usually very effective
- Antimetabolites in case of unresponsiveness to steroids and radiation
- Systemic infliximab: INF1 monoclonal antibody also has promising results

CHAPTER

3

Conjunctiva

COMPETENCY

OP3.1 ELICIT DOCUMENT AND PRESENT AN APPROPRIATE HISTORY IN A PATIENT PRESENTING WITH A “RED EYE” INCLUDING CONGESTION, DISCHARGE, PAIN

SHORT ESSAY

1. A healthcare professional sees a patient with a red eye in the primary healthcare center. Enumerate five differential diagnosis of the same and differentiate among any three of them. (2+3 marks)

Differential Diagnosis for Red Eye

1. Conjunctivitis
2. Uveitis
3. Acute congestive glaucoma
4. Scleritis
5. Endophthalmitis

Differences in Conjunctivitis, Uveitis, and Acute Congestive Glaucoma

Feature	Acute conjunctivitis	Uveitis	Acute congestive glaucoma
Symptoms	Foreign body sensation, lacrimation, discharge, visual acuity is normal	Brow ache, blurred vision	Severe pain, nausea, vomiting, diminished vision
Lids	Edema	Normal	Normal
Conjunctiva	Forniceal and diffuse congestion	Ciliary congestion	Ciliary congestion
Cornea	Usually clear, may have mucous plaques or erosions	KPs may be present	Hazy cornea due to edema
IOP	Normal	Normal, low, or high	Very high, as high as 45 mm Hg
Fundus	Normal	It may be normal or associated with vitritis	Barely visible, the disc is edematous due to high pressure
Treatment	Antibiotics	Steroids and cycloplegics	Peripheral iridotomy, pilocarpine

2. Demonstrate the importance of history taking and examination in the case of a red eye. (5 marks)

- A 'red-eye' can be caused by a variety of conditions ranging from mild to sight-threatening disorders
- Common causes are:
  - Dry eye
  - Conjunctival/corneal foreign body
  - Conjunctivitis
  - Corneal ulcer
  - Iridocyclitis
  - Acute congestive glaucoma
  - Episcleritis
  - Scleritis
  - Endophthalmitis
  - Chemical injuries
  - Trauma to the eye

History Taking

- A thorough history regarding the onset of redness and progression should be taken
- History of injury to the eye or entry of foreign body is important
- The occupation is also important to know as some occupations may predispose to certain conditions

Welders	UV keratopathy
Chemical industries	Chemical injury
Working in extremely hot and dry temperatures	Dry eye

- Associated visual and ocular symptoms should also be elicited in the history

Red-eye with sudden onset pain and photophobia, lacrimation	Probable conjunctival/corneal foreign body iridocyclitis
Red eye with discharge and foreign body sensation	Probable infective conjunctivitis
Red eye with intense itching	Probable allergic conjunctivitis
Red eye with severe pain and decreased vision	Scleritis, endophthalmitis, glaucoma

- Family history may be important in cases of infective etiology
- Use of any drugs also should be asked for

Examination

- The type of congestion is an important part of the examination
- Congestion must be differentiated from conjunctival hemorrhage

Congestion	Conjunctival hemorrhage
Dull red	Bright red (like arterial blood), color may change with time
Diffuse	Localized with sharp borders

- Conjunctival congestion is usually restricted to fornices and palpebral conjunctiva, where ciliary congestion is present around the cornea (circum-corneal congestion)

Causes for conjunctival congestion	Causes for ciliary congestion	Causes for localized congestion	Causes for interpalpebral congestion
• Infective - conjunctivitis • Conjunctival foreign body	• Iridocyclitis • Keratitis • Congestive glaucoma • Endophthalmitis	• Phlyctenulosis • Episcleritis • Scleritis • Conjunctival ulcer	• Dry eye • Allergic conjunctivitis

- A thorough ophthalmic examination should follow anterior segment examination, IOP measurement (if needed), and fundus examination
- Some important points on examination concerning common causes for the red-eye are tabulated below:

Iridocyclitis	KPs, anterior chamber reaction (cells and flare), iris nodules, posterior synechiae, hypopyon
Keratitis	Infiltrates in the cornea, ciliary congestion, hypopyon
Endophthalmitis	Ciliary congestion, lid edema, corneal edema, anterior chamber reaction, hypopyon, yellow reflex in the pupil
Scleritis	Dull red or purplish hue of redness, tenderness with areas of whitening

COMPETENCY

OP3.2 DEMONSTRATE DOCUMENT AND PRESENT THE CORRECT METHOD OF EXAMINATION OF A "RED EYE" INCLUDING VISION ASSESSMENT, CORNEAL LUSTRE, PUPIL ABNORMALITY, CILIARY TENDERNESS

SHORT ESSAY

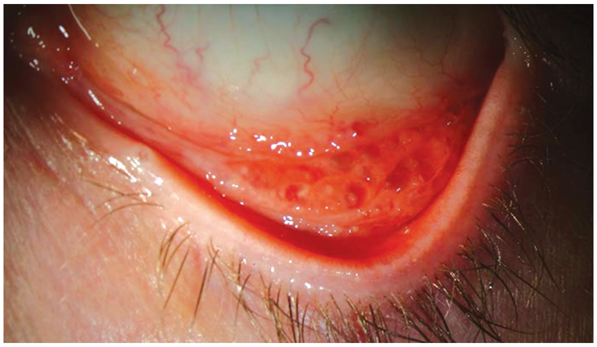
1. Identify the lesion shown in the image. Describe the appearance of papillae and enumerate six causes for the same. (1+1+3 marks)

Lesion is

Conjunctival follicles

Papillae

- Papillae are pale-appearing elevations in the conjunctiva with a central vascular core.
- They occur due to hypertrophic epithelium
- They are seen in the palpebral conjunctiva and limbal area.



Causes

1. Vernal keratoconjunctivitis
2. Trachoma
3. Giant papillary conjunctivitis
4. Atopic conjunctivitis
5. Floppy eyelid syndrome
6. Superior limbic keratoconjunctivitis

SHORT ANSWERS

1. Compare and contrast ciliary congestion and conjunctival congestion. (3 marks)

Feature	Conjunctival congestions	Ciliary congestion
Site of congestion	Palpebral conjunctiva > bulbar conjunctiva	Around the cornea
Source of congestion	Conjunctival vessels	Anterior ciliary vessels
Causes	Conjunctivitis Conjunctival foreign bodies	Iridocyclitis Corneal ulcer Acute congestive glaucoma
Effect of topical phenylephrine	Congestion blanches	Congestion does not blanch as it is deep

2. Describe the appearance of follicles and enumerate three causes. (2+1 marks)

Follicles

Appearance

- Appear as rounded tiny elevations with surrounding vasculature, more in the fornices
- They appear as sago grains with blood vessels in the periphery

Causes

1. Viral conjunctivitis
2. Trachoma
3. Ophthalmia nodosa

3. Explain the appearance of subconjunctival scarring and give two causes. (2+1 marks)

Subconjunctival Scarring

Appearance

Seen as a whitish-gray area under the conjunctiva while the lids are everted

Causes

1. Trachoma
2. Stevens–Johnson syndrome

4. Elicit ciliary tenderness and explain its importance. (2+1=3 marks)

Ciliary Tenderness Elicitation

- Ciliary tenderness is a clinical sign that is elicited by pressing gently in the ciliary area (2 mm behind the limbus)
- The superior limbus is chosen due to convenience
- The examiner uses her thumb to gently press on the area upper ciliary area through the upper lids
- The patient winces in response

Importance

It is typically seen in acute iridocyclitis and is helpful when the anterior chamber reaction is too subtle for diagnosis

5. Enumerate four causes of the conjunctival membrane and explain how it is managed. (2+1 marks)

Conjunctival Membrane

- It is an aggregation of inflammatory exudates that get organized on the palpebral conjunctiva
- It appears dull gray and bleeds when peeled. It is a sign of severe inflammation

Causes

1. Diphtheritic conjunctivitis (classically described)
2. Viral conjunctivitis
3. Gonococcal conjunctivitis
4. Ligneous conjunctivitis
5. Steven–Johnson syndrome

Management

- It is treated with topical antibiotics and lubricants
- A true membrane should not be peeled as it can lead to symblepharon
- The pseudomembrane is peeled under visualization through a slit lamp

COMPETENCY

OP3.3 DESCRIBE THE AETIOLOGY, PATHOPHYSIOLOGY, OCULAR FEATURES, DIFFERENTIAL DIAGNOSIS, COMPLICATIONS. AND MANAGEMENT OF VARIOUS CAUSES OF CONJUNCTIVITIS

LONG ESSAYS

1. A family of four presents with a history of red eye and watering that started first in the father followed by the mother and then her two children. On examination, there is severe conjunctival congestion with petechial hemorrhages in both eyes. All of them have tender preauricular lymphadenopathy and there is a serous and occasional mucoid discharge.

A. State the most probable diagnosis. (1 mark)
B. Name four causative organisms for this condition. (4 marks)
C. Justify your diagnosis. (2 marks)
D. Describe the management of this condition. (3 marks)
E. Contrast between the clinical presentation of bacterial and viral inflammation. (5 marks)

A. Most Probable Diagnosis

Viral conjunctivitis.

B. Causes

1. Adenovirus
2. Herpes virus
3. Coxsackievirus
4. Enterovirus

C. Points in Favor of Viral Conjunctivitis

- History of rapid transmissibility from one person to another
- History of red eye and watering
- Petechial hemorrhages in the conjunctiva
- Preauricular lymphadenopathy

D. Management

- It is mainly supportive as the infection is self-subsiding
- Topical lubricants and broad-spectrum antibiotics are given for a week
- The patients are instructed to prevent the further spread of infection by using separate face towels and avoiding contact with other individuals

E. Differences between Bacterial and Viral Conjunctivitis

Feature	Viral conjunctivitis	Bacterial conjunctivitis
Symptoms	Serous discharge	Mucoid discharge
Conjunctival signs	Higher incidence of petechial hemorrhages, pseudo-membrane, and chemosis Lid edema tends to be more severe Follicles appear more commonly	Congestion of conjunctiva A lesser degree of lid edema, hemorrhages, chemosis, and membranes. No follicles
Preauricular lymphadenopathy	Higher incidence	Lower incidence
Response to topical antibiotics	Lesser	Faster
Transmissibility	Higher	Lower
Duration of symptoms	Longer, but self-subsiding	Shorter
Systemic associations	It may be associated with fever or pharyngitis	Only some types of bacterial conjunctivitis have systemic associations: Otitis media: in children Gonorrhea: hyperacute conjunctivitis

2. A 5-day-old child presents with watering and redness from both eyes associated with mucopurulent discharge since 1 day. There is mild lid edema, and congestion of the palperbral conjunctiva.

A. Name the condition. (0.5 mark)

B. Which organism is the probable cause for this? (0.5 mark)

C. Compare different etiological agents responsible for this condition and their clinical features. (6 marks)

D. Explain the management of this condition. (3 marks)

A. This Condition is

Ophthalmia neonatorum

B. Most Probable Cause

Staphylococcus aureus

C. Etiological Agents vis-à-vis Clinical Features

Feature	Etiology	Incubation period	Features
Chemical conjunctivitis	Application of silver nitrate	4–6 hours	Serous discharge
Gonococcal conjunctivitis	*N. gonorrhoeae*	2–4 days	Purulent discharge Lid edema Hyperemia
Bacterial conjunctivitis	*Streptococcus* *Staphylococcus*	4–5 days	Mucoid discharge
Neonatal inclusion conjunctivitis	*Chlamydia oculogenitalis*	5–14 days	Mucoid discharge
Herpetic conjunctivitis	Herpes simplex	5–7 days	Serous discharge

D. Management of Ophthalmia Neonatorum

Feature	Treatment
Chemical conjunctivitis	Self-limiting
Gonococcal conjunctivitis	Bacitracin ointment, topical atropine if the cornea is involved
	Systemic treatment
	• Ceftriaxone 75–100 mg/kg/day IV or IM, QID
	• Cefotaxime 100–150 mg/kg/day IV or IM, 12 hourly
	• Ciprofloxacin 10–20 mg/kg/day or Norfloxacin 10 mg/kg/day
Bacterial conjunctivitis	Broad-spectrum antibiotics
Neonatal inclusion conjunctivitis	Topical tetracycline 1% or erythromycin 0.5% eye ointment QID for 3 weeks. Systemic erythromycin 125 mg orally, QID for 3 weeks
Herpetic conjunctivitis	Topical antivirals

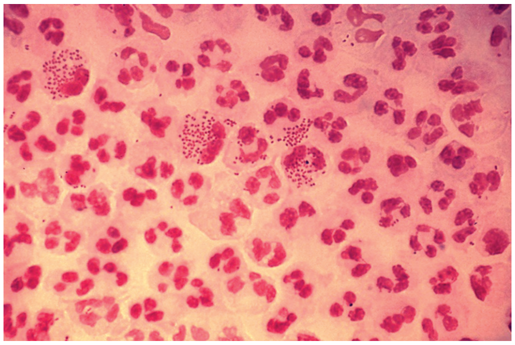
SHORT ESSAYS

1. A conjunctival swab from a patient with conjunctivitis shows the following picture.

Explain the clinical features of the condition caused by this organism.

Or

Appraise the clinical features of acute gonococcal conjunctivitis. (5 marks)



- The image shows gram-negative diplococci suggestive of *Neisseria gonorroheae*
- Gonococcal conjunctivitis is also known as Hyperacute conjunctivitis and is caused by *N. gonorrhoeae*

Clinical Features

It usually follows genital infection and thus is more on the right side owing to an increased number of individuals being right-handed

Symptoms

- Hyperacute presentation
- Purulent discharge
- Redness
- Severe eye pain

Signs

- Severe lid edema with tenderness
- Velvety red conjunctiva with chemosis
- A membrane may be present
- Tender preauricular lymph nodes
- Profuse discharge

Complications

1. *N. gonorrhoeae* can invade intact epithelium and thus there is a risk of corneal ulcer formation, perforation
2. Endophthalmitis

2. Describe the clinical features and management of acute mucopurulent conjunctivitis. (3+2 marks)

Acute Mucopurulent Conjunctivitis

- It is the most common form of conjunctivitis and is caused by gram-positive bacteria like *Staphylococcus aureus*, *Staphylococcus epidermidis*, and *Streptococcus pneumoniae*
- It is also known as acute catarrhal conjunctivitis
- It spreads via contaminated fingers and may accompany measles and scarlet fever

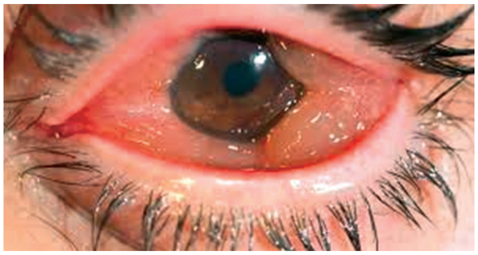
Clinical Features

Symptoms

- Redness
- Watering
- Foreign body sensation
- Mucoid or sometimes purulent discharge
- Rarely colored halos due to mucoid flakes

Signs

- Lid edema
- Conjunctival congestion
- Mucoid discharge in fornices or lid margins
- The cornea is usually unaffected



3. Identify the pathology in the conjunctiva. Enumerate four causes of membranous conjunctivitis. Elaborate the management of the same. (1+2+2 marks)

Pathology is

Conjunctival chemosis

Causes of Membranous Conjunctivitis

1. *Corynebacterium diphtheriae*
2. *Streptococcus hemolyticus*
3. Viral conjunctivitis
4. Ligneous conjunctivitis

Management

- Treatment of infectious membranous conjunctivitis involves the use of topical antibiotics and lubricants
- Confirmed cases of diphtheria (rare) should receive
    - Penicillin eye drops (1:10000 units per ml) to be instilled every half-hourly
    - Antidiphtheric serum (ADS) should be instilled every hour
    - Broad-spectrum antibiotic ointment should be applied at bedtime
    - Systemic therapy: Crystalline penicillin 5 lac units should be injected intramuscularly twice a day for 10 days and ADS: (50 thousand units) should be given intramuscularly stat
- Ligneous conjunctivitis is treated with topical plasminogen or topical fresh frozen plasma

4. Summarise the etiology, clinical features, and management of angular conjunctivitis. (1+2+2 marks)

Angular Conjunctivitis

It is a type of chronic conjunctivitis, in which there is a mild inflammation of the conjunctiva and lid margins at the medial and lateral canthi.

Etiology

- Causative organism: *Moraxella axenfeld* (diplobacillus)
- Predisposing factor: Chronic conjunctivitis
- Organisms usually reside in the nasal cavity and are transmitted via finger contamination
- The causative organism produces a proteolytic enzyme that macerates the epithelium. This enzyme collects at the angles by the action of tears and thus signs are seen at the angles

Clinical Features

Symptoms

- Tearing and foreign body sensation
- Redness at the sides of the eye

Signs

- Congestion of conjunctiva at the canthi
- Lid margin congestion
- Erythema at the skin of canthi
- Maceration of skin

Management

- Oxytetracycline (1%) eye ointment 2–3 times a day for 2 weeks
- Zinc lotion instilled in daytime and zinc oxide ointment at bedtime inhibits the proteolytic enzyme

5. Enumerate four viruses that commonly cause conjunctivitis. Explain the manifestations of pharyngoconjunctival fever and epidemic keratoconjunctivitis. (2+3 marks)

Causes of Viral Conjunctivitis

1. Adenovirus
2. Herpes simplex virus
3. Picorna virus
4. Poxvirus

Pharyngoconjunctival Fever Manifestations

It is an adenoviral infection caused by subtypes 3 and 7.

Symptoms

- It is characterized by a triad of symptoms: acute follicular conjunctivitis, pharyngitis, fever, and preauricular lymphadenopathy
- The disease primarily affects children and appears in epidemic form

Signs

- Conjunctiva is congested, may be associated with petechial hemorrhages
- Follicular reaction
- The cornea is clear

Epidemic Keratoconjunctivitis Manifestations

- It is an adenoviral infection caused by serotypes 8 and 19, occurs in epidemics, and can cause punctate keratitis
- The infection spreads through contaminated fingers and instruments.

Symptoms

- The incubation period of 8 days
- Redness of eyes
- Lacrimation
- Foreign body sensation
- Photophobia (due to corneal involvement)

Signs

- Hyperemia of the conjunctiva, with petechial hemorrhages
- Chemosis and follicles
- Pseudomembranes
- Preauricular lymphadenopathy
- Corneal involvement: Superficial punctate keratitis, subepithelial infiltrates, epithelial microcystic lesions

6. What is Apollo conjunctivitis and why is it named so? Describe its clinical features. (1+1+3 marks)

Apollo Conjunctivitis

- It is an another name for acute viral hemorrhagic conjunctivitis that caused an epidemic in the year 1969
- It was first seen after the Apollo XI spacecraft was launched and hence is sometimes known as ‘Apollo conjunctivitis’

Clinical Features

The disease is extremely contagious and is transmitted by direct hand-to-eye contact.

Symptoms

- Redness
- Watering
- Pain

Signs

- Lid edema
- Punctate hemorrhages in palpebral conjunctiva, initially
- In severe cases, hemolacria may occur (bloody tears)
- Follicles may be present

7. Enumerate two viruses implicated in pandemics that have conjunctival manifestations. Describe the conjunctival manifestations of COVID-19. Demonstrate the knowledge of its epidemiological relevance. (1+2+2 marks)

Viruses Implicated In Pandemics That Have Conjunctival Manifestations

1. SARS-CoV
2. Influenza virus

Conjunctival Manifestations of COVID-19

- COVID-19 is the present-day pandemic with various manifestations, mainly being fever and respiratory infection
- It has a conjunctival manifestation in the form of conjunctivitis
- The main symptoms are red eye, foreign body sensation, lacrimation, serous discharge, and signs that include follicular conjunctivitis
- It may be the only sign of corona virus infection
- Conjunctivitis resolves by itself and is treated with broad-spectrum antibiotics

Epidemiological Relevance

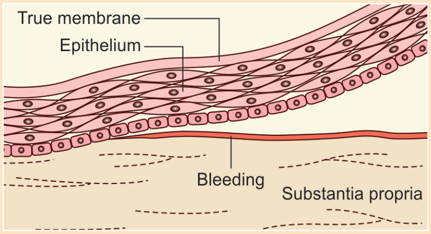
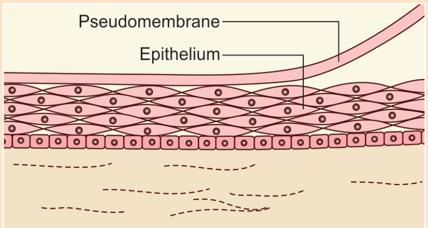
- Its importance lies in the identification of coronavirus infection.
- It can be diagnosed by the conjunctival and nasopharyngeal swab
- The risk of transmission through tears is low
- Any case of viral conjunctivitis should be viewed with a suspicion of COVID-19 infection, especially during the period of the pandemic and if there is a history of contact with a patient with the same

8. Compare and contrast true membrane and a pseudomembrane. (5 marks)

True Membrane and Pseudomembrane

Feature	True membrane	Pseudomembrane
Cause	Diphtheritic conjunctivitis Beta hemolytic *Streptococcus*	*Corynebacterium diphtheriae* of low virulence, staphylococci, streptococci, *H. influenzae*, and *N. gonorrhea*. Herpes simplex and adenoviral epidemic keratoconjunctivitis
Clinical features	Bleeds on peeling Leaves a raw surface	Does not bleed on peeling Leaves intact epithelium

Contd.

Feature	True membrane	Pseudomembrane
Pathology	Organized fibrinous exudates during inflammation adhered to the epithelium (Fig. 3.3.1)  True membrane Epithelium Bleeding Substantia propria **Fig. 3.3.1:** True membrane	Organized exudates not adhered to the epithelium (Fig. 3.3.2)  **Fig. 3.3.2:** Pseudomembrane
Treatment	Antidiphtheritic treatment	Peeling of pseudomembrane with supportive antibiotics

SHORT ANSWERS

1. Appraise the clinical features of acute hemorrhagic conjunctivitis. (3 marks)

Acute Hemorrhagic Conjunctivitis

- It is hyperacute conjunctivitis characterized by multiple conjunctival hemorrhages, conjunctival hyperemia, and mild follicular hyperplasia
- It is usually associated with infection due to picornaviruses (enterovirus type 70)
- The disease is extremely contagious and is transmitted by direct hand-to-eye contact

Clinical Features

Symptoms

- Redness
- Watering
- Pain

Signs

- Lid edema
- Punctate hemorrhages in palpebral conjunctiva, initially
- In severe cases, hemolacria may occur (bloody tears)
- Follicles may be present

2. Describe Perinaud's oculoglandular syndrome. Enumerate two causative agents. (1+2 marks)

Perinaud's Oculoglandular Syndrome

- It is a rare unilateral granulomatous conjunctivitis
- Characterized by follicular inflammation of the conjunctiva accompanied by preauricular lymphadenopathy and fever

Causes

1. Tularemia
2. Cat scratch disease

COMPETENCY

OP3.4 DESCRIBE THE AETIOLOGY, PATHOPHYSIOLOGY, OCULAR FEATURES, DIFFERENTIAL DIAGNOSIS, COMPLICATIONS AND MANAGEMENT OF TRACHOMA

LONG ESSAYS

1. A 45-year-old man hailing from Bihar came for regular follow-up. He has trichiatic lashes in his lower lids of both eyes and subconjunctival fibrosis. In the cornea, there is xerosis and diffuse fluorescein staining. The superior portion of the cornea shows opacification.

A. Identify the probable infectious cause for this condition of the eye.	**(1 mark)**
B. Where is this disease endemic?	**(1 mark)**
C. Describe the epidemiology of this disease.	**(5 marks)**
D. Discuss the clinical features of the acute form of this disease.	**(5 marks)**
E. Describe the management of present condition.	**(3 marks)**

A. Most Probable Infectious Cause for this Condition is

Trachoma

B. Endemic in

- Parts of North Africa
- Also seen in many parts of India

C. Epidemiology of Trachoma

Etiology

Causative agent is *Chlamydia trachomatis* serotypes A, B, Ba, C

Source of Infection

- The affected individual is a source of infection
- The tears and conjunctival discharge are a source of infection

Mode of Transmission

- Direct spread by inoculation through contaminated fingers
- Vector-borne transmission is through flies
- Fomite transfer plays an important role: Use of common towels, handkerchiefs, contaminated tonometers, beddings, etc.

Host Factors

- Primary infection is more common in early childhood
- Poor socioeconomic conditions are associated with a higher incidence of trachoma

Environmental Factors

- Dry, dusty climates
- Overcrowding, poor environmental sanitation, the paucity of clean drinking water increase the incidence of trachoma
- Exposure to dust, chemicals, and irritants increases the risk of transmission

Incubation Period

- 8–10 days

D. Clinical Features of Active Trachoma

Symptoms

- The primary trachoma is usually silent
- When there is a superinfection, it presents with usual symptoms of conjunctivitis
- 'Trachoma dubium': Doubtful trachoma

Signs

Conjunctival signs
- Congestion of upper palpebral conjunctiva
- Follicles in the upper palpebral as well as bulbar conjunctiva
- Papillary hypertrophy
- Subconjunctival scarring (Artl's line)
- Concretions: due to inspissated mucous cells in glands of Henle

Corneal signs
- Herbert follicles: At the limbus
- Herbert's pits: Due to necrosis of follicles
- Pannus: Infiltration into the cornea associated with vascularization is seen in the upper part. It can be progressive (when vascularization precedes infiltration) or regressive (vascularization lags)
- Superficial ulcers may develop at the pannus. They tend to be chronic and indolent
- Corneal opacity in later stages

E. Management

- Presently, the patient is in the stage of sequelae
- The problems to be addressed are dry eye and entropion with trichiasis
- As the opacification in the upper part of the cornea and not covering the pupil, keratoplasty is not indicated

Treatment of Dry Eye

- Mild forms of dry eye are treated with tear film substitutes
- Severe dry eye may be treated with punctal occlusion by using silicone plugs

Entropion and Trichiasis

- Trichiasis may be temporarily treated with epilation. But there is recurrence
- It can also be treated with cryotherapy or electrolysis
- Bilamellar tarsal resection is the surgical procedure of choice
- Entropion needs corrective surgery in the form of tarsal wedge resection, anterior lamellar resection, and transposition of the tarsoconjunctival wedge

2. *Chlamydia* is an obligate intracellular organism.

A. Summarize ocular infections caused by *Chlamydia*. (3 marks)
B. Explain McCallan's and the WHO classification of trachoma. (4 marks)
C. Explain the steps taken by the WHO in the eradication of trachoma. (3 marks)

A. Ocular Infections Caused by *Chlamydia*

1. **Acute chlamydial conjunctivitis and neonatal inclusion conjunctivitis**
   - Also known as paratrachoma, It is caused by serotypes D to K

- It is an oculogenital infection, spreads to the eye by finger transmission from genitals and from one eye to another through secretions
- In males, it causes nonspecific urethritis and in females, it causes dysuria, pelvic inflammatory disease, and salpingitis
- Symptoms
    - Subacute presentation
    - Redness
    - Watering
    - Foreign body sensation
- Signs
    - Serous/mucopurulent discharge
    - Follicles in the lower fornix
    - Tender preauricular lymph nodes
    - Peripheral corneal infiltrates
    - Subconjunctival scarring

2. **Trachoma (Egyptian blindness)**
    - It is an infection caused by serotypes A, B, Ba, and C
    - The infection tends to be self-limiting but has recurrences
    - The problem is resultant scarring which leads to trichiasis and thus causing corneal complications and opacification
    - Thus, the disease should be recognized early and treated
    - It is hyperendemic and is more commonly seen in north Africa, the Middle East, and southeast Asia where the climate is dry and dusty.
    - It is responsible for around 20% of the world's blindness
3. **Lymphogranuloma Venerum Conjunctivitis**
    - It is caused by serotypes L1, L2, and L3
    - Its manifestations are similar to adult inclusion conjunctivitis

B. Classification of Trachoma

McCallan's Classification

1. **Incipient trachoma or stage of infiltration**
    - Hyperaemia of palpebral conjunctiva and immature follicles
2. **Established trachoma or stage of florid infiltration**
    - Appearance of mature follicles, papillae, and progressive corneal pannus
        - Follicle predominant
        - Papillary predominant
3. **Cicatrizing trachoma or stage of scarring**
    - Scarring of the palpebral conjunctiva
4. **Healed trachoma or stage of sequelae**
    - Sequelae due to cicatrization give rise to symptoms

WHO Classification

F	• (TF) Follicular stage: active inflammation with follicular hypertrophy. • Diagnosis: ≥5 follicles (≥0.5 mm diameter) must be present on the upper tarsal conjunctiva. The deep tarsal vessels should be visible through the follicles and papillae
I	• (TI) Intense inflammation: Inflammatory thickening in the upper tarsal conjunctiva obscuring more than half of vessels
S	• (TS)Trachomatous scarring: visible as whitish area of scarring in the palpebral conjunctiva
T	• (TT)trichiasis is defined when atleast one eyelash is touching the cornea
O	• (TO) opacity: easily visible opacity in the pupillary area. the opacity should be so dense that a part of pupillary margin is not visible. (leukomatous) vision <6/18

C. WHO in the Eradication of Trachoma

- World Health Organization has launched a GET 2020 (Global Eradication of Trachoma by 2020) and adopted a SAFE strategy

S	Surgery (tertiary prevention)	• WHO recommends bilamellar tarsal rotation surgery for all affected persons (with trichiasis and entropion)
A	Antibiotics use (secondary prevention)	• In areas with >10% of the population with active trachoma • Oral azithromycin (20 mg/kg up to 1g) to be given to all individuals of the community • Topical tetracycline ointment twice daily for 6 weeks for all pregnant women, <6 months children, and individuals allergic to macrolides • In areas 5–10% prevalence of trachoma • Antibiotic therapy as described above for family members and close contacts of the patients • In areas with prevalence <5% of trachoma • Antibiotic treatment only to patients
F	Facial hygiene (primary prevention)	• Frequent face wash with clean water • Avoidance of using common towels and handkerchiefs
E	Environmental changes (primordial prevention)	• Environmental sanitation • Provision of good water supply • Improved toilets • Control of flies by using insecticide-sprays • Health education • Keeping animal pens away from human household

SHORT ESSAYS

1. Summmarise the management of cicatricial trachoma. (5 marks)

Treatment of Dry Eye

- Mild forms of dry eye are treated with tear film substitutes
- Severe dry eye may be treated with punctal occlusion by using silicone plugs

Entropion and Trichiasis

- Trichiasis may be temporarily treated with epilation
- But there is recurrence
- It can also be treated with cryotherapy or electrolysis

- Bilamellar tarsal resection is the surgical procedure of choice
- Entropion needs corrective surgery in the form of tarsal wedge resection, anterior lamellar resection, and transposition of the tarso-conjunctival wedge

Treatment of Corneal Opacity

- Penetrating keratoplasty is performed in cases of corneal opacity
- Keratoprosthesis is advocated for corneal blindness with severe limbal cell deficiency owing to loss of goblet cells

Others

In presence of concretions, they are removed with a hypodermic needle under topical anesthesia.

2. Describe the etiology, clinical features, and management of adult inclusion conjunctivitis. (1+2+2 marks)

Adult Inclusion Conjunctivitis

Etiology

- Adult inclusion conjunctivitis is caused by *Chlamydia trachomatis*, serotypes D to K
- It is an oculogenital infection, spreads to the eye by finger transmission from genitals and from one eye to another through secretions
- In males, it causes nonspecific urethritis and in females, it causes dysuria, pelvic inflammatory disease, and salpingitis

Clinical Features

Symptoms

- Subacute presentation
- Redness
- Watering
- Foreign body sensation

Signs

- Serous/mucopurulent discharge
- Follicles in the lower fornix
- Tender preauricular lymph nodes
- Peripheral corneal infiltrates
- Subconjunctival scarring

Management

Investigations

- Conjunctival smear for PCR, direct monoclonal fluorescent antibody test, McCoy cell culture
- Rule out genital disease

Treatment

- Tetracycline eye ointment twice a day for 10 days
- Oral azithromycin 1 g or oral doxycycline 100 mg twice a day for 10 days

3. Tabulate features of cicatricial trachoma. (5 marks)

Cicatricial Trachoma

Conjunctival manifestations	• Subconjunctival scarring is visible as greyish white linear bands below the mucosa of the conjunctiva • Concretions are hard inspissated mucus that is present in the depressions of glands of Henle • Xerosis • Symblepharon
Corneal manifestations	• Superficial punctate erosions owing to trichiasis • Regressive pannus is commonly seen in cicatricial trachoma • Herbert's pits: Circular or oval pits that are formed due to the healing of Herbert's follicles • Superior corneal opacity may involve the pupil • Corneal xerosis and nonhealing epithelial defects owing to limbal stem cell deficiency
Lid signs	• Entropion • Trichiasis • Tylosis • Madarosis
Lacrimal apparatus	• Chronic dacryocystitis • Chronic dacryoadenitis

SHORT ANSWERS

1. Explain the term trachoma dubium. (3 marks)

- Trachoma dubium means 'doubtful trachoma'
- This term is applied for primary trachoma as it has very mild symptoms like lacrimation, mild foreign body sensation, and mild scanty mucoid discharge
- As these symptoms are very nonspecific, primary infection of trachoma is usually missed
- It becomes evident only if there is a superadded bacterial infection

2. Enumerate infections caused by the TRIC agents. (3 marks)

- TRIC agents refer to serotypes of *Chlamydia* from A to K causing trachoma and inclusion conjunctivitis
  1. Hyperendemic trachoma
  2. Paratrachoma—adult and neonatal inclusion conjunctivitis

3. Explain the corneal manifestations of trachoma. (3 marks)

Corneal Manifestations in Active Trachoma

- Herbert follicles: At the limbus
- Pannus: infiltration into the cornea associated with vascularization is seen in the upper part. It can be progressive (when vascularization precedes infiltration) or regressive (vascularization lags)
- Superficial ulcers may develop at the pannus, they tend to be chronic and indolent

Corneal Manifestations in Cicatricial Trachoma

- Superficial punctate erosions owing to trichiasis
- Regressive pannus is commonly seen in cicatricial trachoma
- Herbert's pits: Circular or oval pits that are formed due to the healing of Herbert's follicles

- Superior corneal opacity may involve the pupil
- Corneal xerosis and nonhealing epithelial defects owing to limbal stem cell deficiency

4. Describe the laboratory diagnosis of trachoma. (3 marks)

Laboratory diagnosis in the form of advanced laboratory tests is employed for research purposes only, as the diagnosis of trachoma is mainly clinical

1. Conjunctival cytology
    - Study of scrapings from the conjunctiva show HP (Halberstädter Prowazek) bodies when stained with Giemsa stain
    - Multinucleated giant cells (Leber cells) may be seen in Giemsa smears
2. ELISA for detection of *Chlamydia* antibodies, PCR, microimmunofluorescence
3. Isolation of *Chlamydia* is possible by the yolk sac inoculation method and tissue culture technique. Standard single-passage McCoy cell culture requires at least 3 days
4. Serotyping is done by detecting antibodies by microimmunofluorescence

5. Explain the terms

A. Herbert's pits (1 mark)
B. Artl's line (1 mark)
C. Pannus (1 mark)

A. Herbert's pits

- When the follicles along the limbus heal, they form depressions called Herbert's pits
- It is classically seen in cicatricial trachoma

B. Artl's line

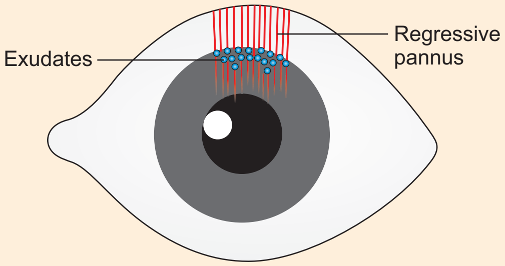
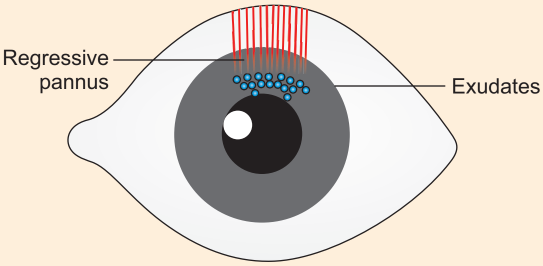
- Greyish-white linear subconjunctival scars that are seen in the palpebral conjunctiva
- They are seen in cicatricial trachoma

C. Pannus

- It is the infiltration into the cornea associated with vascularization and is seen in the upper part
- Progressive pannus is seen in active trachoma, where the infiltration precedes vascularization
- Regressive pannus is seen in cicatricial trachoma. In this, the infiltrates recedes vascularization

6. Compare and contrast between progressive and regressive pannus. (5 marks)

Progressive and Regressive Pannus

Progressive pannus (Fig. 3.4.1)	**Regressive pannus (Fig. 3.4.2)**
• Vascularization recedes infiltration • Seen in active trachoma	• Infiltration recedes vascularization • Seen in cicatricial trachoma
 **Fig. 3.4.1:** Progressive pannus	 **Fig. 3.4.2:** Regressive pannus

COMPETENCY

OP3.5 DESCRIBE THE AETIOLOGY, PATHOPHYSIOLOGY, OCULAR FEATURES, DIFFERENTIAL DIAGNOSIS, COMPLICATIONS AND MANAGEMENT OF VERNAL CATARRH

LONG ESSAY

1. **A 9-year-old boy presents with severe itching in both eyes for 3 weeks. He complains of exacerbation of symptoms during the summer season. On examination, the following image is seen.**

   A. State the probable diagnosis. (1 mark)

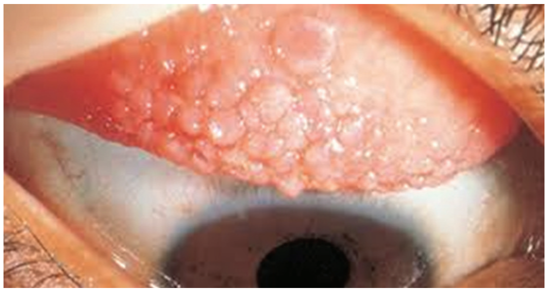
   B. Enumerate the clinical features of this disease. (5 marks)

   C. Explain the management of this condition. (3 marks)

   D. Name 1 complication. (1 mark)

A. Probable Diagnosis

Vernal keratoconjunctivitis (VKC)



B. Clinical Features

Symptoms

- Recurrent episodes of bilateral itching
- More in boys, and more in summer seasons
- Associated with stringy discharge

Signs

Conjunctival form or palpebral form	• Micropapillae, macropapillae in upper palpebral conjunctiva • Dusky congestion of the conjunctiva • Ropy discharge • Gelatinous thickening at the limbus • Discrete raised whitish dots at limbus: Tranta's spots
Corneal predominance	• Punctate erosions: Very common • Epithelial macroerosion • Shield ulcer: Shallow transverse ulcer with clean margins and base • Vernal plaque due to coating of the macro-erosion with exudation • Subepithelial scarring • Pseudogerontoxon: Localized area resembling arcus senilis occurs following healing of inflamed segment
Mixed variant	• Has features of both conjunctival and corneal involvement

C. Management

1. **Local therapy**
   - Topical steroids
     - It is the mainstay of treatment for VKC, but one must be careful not to miss steroid-induced glaucoma, and hence it must be tapered when the disease is controlled
     - The drugs usually used are prednisolone and dexamethasone
     - In severe cases, fluorometholone, medrysone, and in mild cases loteprednol are used
     - Frequent instillation up to 4th hourly for first five days followed by four times a day application to be continued for 3 weeks

    - IOP should be carefully monitored because of corneal erosions, and fundus examination every 2 weeks for glaucomatous changes should be done
  - Mast cell stabilizers
    - Slightly weaker in action than steroids, nonetheless, have an important role in stabilizing the disease
    - Their action takes longer to begin and hence should be continued for a month following the onset of the disease
    - Examples are sodium cromoglycate (2%) and olopatadine (0.05% and 0.1%)
  - Mucolytics like acetylcysteine (0.5%)
    - Useful in the treatment of early plaque formation
  - Topical cyclosporine (1%) drops
    - Can be used if steroids are contraindicated or the disease is not controlled by steroids

2. **Systemic therapy**
   - Oral antihistaminics in severe cases
   - Oral steroids for a short duration for very severe, nonresponsive cases
   - Treatment of large papillae: Supratarsal injection of steroid or cryotherapy can be tried
3. **Treatment of vernal keratopathy**
   - Steroids are the mainstay of treatment
   - The corneal epithelial loss is a result of immune-mediated destruction and sometimes due to papillae
     - *Punctate epithelial keratitis:* Steroids
     - *Vernal plaque:* Acetylcysteine, surgical excision by superficial keratectomy
     - *Shield ulcer:* Steroids, surgical treatment in the form of debridement, superficial keratectomy, excimer laser therapeutic keratectomy as well as amniotic membrane transplantation to enhance re-epithelialization

D. Complication

Shield ulcer

SHORT ESSAYS

1. A 33-year-old woman was brought to the emergency with a history of vesiculopapular lesions on the skin and mucosa following ingestion of phenytoin.

A. What is the most probable diagnosis? (1 mark)

B. Describe the ocular manifestations of the aforementioned condition. (1 mark)

C. Explain the management in the acute phase and cicatricial phase. (2 marks)

D. Name another dermatological disorder leading to similar manifestations. (1 mark)

A. The Most Probable Diagnosis is

- Stevens–Johnson syndrome
- It is characterized by hypersensitivity to a drug or a microorganism
- As a result, there are erosions in the mucosa and vesiculopapular lesions on the skin throughout the body

B. Ocular Manifestations in the Active Inflammation Stage Include

- Intense congestion of the conjunctiva
- Erosions in conjunctiva and cornea

- Mucoid discharge
- Loss of limbal stem cells
- As a consequence, these individuals develop severe dry eyes, nonhealing corneal ulcers, entropion, symblepharon, trichiasis, and limbal stem cell deficiency

C. Management

In Acute Phase

- Ocular care includes copious use of topical steroids to decrease the ongoing inflammation, tear film substitutes, and antibiotics
- If there are associated corneal erosions, patching the eye with ointment or bandage contact lens can be used.
- A symblepharon shell or a scleral contact lens may be placed to keep the bulbar and the palpebral conjunctiva separate
- Forniceal sweeping is done twice a day to break if any symblepharon has developed. It is done under topical anesthesia with a glass rod coated with a lubricating ointment

In Cicatricial Phase

- Dry eyes are treated with tear film substitutes
- As the condition is bilateral, there is bilateral stem cell deficiency due to destruction of the limbal stem cells
  - Recurrent corneal erosions may be treated with bandage contact lenses and amniotic membrane
  - Limbal stem transfer is necessary in severe cases

D. Another Dermatological Disorder Leading to Similar Manifestations

Ocular cicatricial pemphigoid

2. Classify allergic disorders of the conjunctiva and explain common clinical features of all the types. (2+3 marks)

Allergic Disorders of the Conjunctiva

1. Acute/subacute allergic conjunctivitis
2. Phlyctenular keratoconjunctivitis
3. Vernal keratoconjunctivitis
4. Atopic keratoconjunctivitis
5. Giant papillary conjunctivitis

Common Clinical Features

Symptoms

- The main symptom of allergic conjunctivitis is itching of the eyes
- It is usually bilateral, chronic, and episodic
- Allergy tends to be chronic and recurrent
- It is associated with itching more at the medial canthus due to the pooling of tears and increased concentration at the lacus lacrimalis
- It may be associated with dry eye and cause foreign body sensations
- Some types of allergy have a systemic association (atopy). So, they may have allergic manifestations in the skin also

Signs

- Acute allergy presents with conjunctival congestion, chemosis, and lid edema
- Chronic allergy is characterized by the formation of papillae (hypertrophy of the epithelium) in response to the allergen
- The congestion in chronic allergy tends to be dusky and restricted to the exposed parts of the conjunctiva
- Some types of allergic conjunctivitis have corneal involvement and thus have erosions or epithelial defects
- They all respond well to topical mast cell stabilizers and steroids

3. Describe vernal keratopathy—its features and management. (3+2 marks)

Vernal Keratopathy

Vernal keratoconjunctivitis may present with predominantly corneal manifestations and is termed vernal keratopathy.

Clinical Features

- Punctate corneal erosions
- Epithelial macroerosion
- Shield ulcer: Shallow transverse ulcer with clean margins and base
- Vernal plaque due to coating of the macroerosion with exudation
- Subepithelial scarring
- Pseudogerontoxon: Localized area resembling arcus senilis occurs following healing of inflamed segment

Management

- Topical steroids are the mainstay of treatment
- The corneal epithelial loss is a result of immune-mediated destruction and sometimes due to papillae
  - Punctate epithelial keratitis: Steroids
  - Vernal plaque: Acetylcysteine, surgical excision by superficial keratectomy
  - Shield ulcer: Steroids, surgical treatment in the form of debridement, superficial keratectomy, excimer laser therapeutic keratectomy as well as amniotic membrane transplantation to enhance re-epithelialization

4. Describe the etiology and clinical features of atopic keratoconjunctivitis. (2+3 marks)

Atopic Keratoconjunctivitis

Etiology

- It is associated with strong family history
- There is usually associated atopy
- Types 1 and 4 hypersensitivity reactions to the allergen cause inflammatory changes in the skin and the conjunctiva and the process is usually chronic
- There are mast cells and eosinophils in the conjunctiva

Clinical Features

Symptoms

- The disease has a chronic course and is seen inolder individuals
- Episodes of itching
- Lacrimation
- Burning sensation
- Redness
- Itching and lesions in the skin especially in flexures

Signs

- Tylosis, rounded lid margins
- Papillae, conjunctival scarring
- Conjunctival congestion, chemosis
- Corneal punctate erosions, more in lower half, vascularization, plaques
- Shortening of lid, symblepharon

5. Create a stepwise management algorithm for vernal keratoconjunctivitis. (5 marks)

Vernal keratoconjunctivitis is clinically graded as mild, moderate, severe, and blinding disease depending on clinical signs like papillae, Horner–Trantas spots, superficial punctate erosions, annular limbal inflammation, limbal stem deficiency, and shield ulcer.

Mild disease	Moderate disease	Severe disease	Blinding disease
Symptoms + papillae	Mild + Horner–Trantas spots + <6 hours of limbal thickening	Moderate + >6 hours of limbal thickening + macroerosions + pannus	Severe + shild ulcer + vascularization of cornea + limbal stem cell deficiency
Avoidance of the known allergen	Treatment for mild disease	Treatment for moderate disease	Treatment for severe disease
Lubricants	+	+	+
Antihistaminics and mast cell stabilizers	Loteprednol Cyclosporine 0.5% and 1%	Potent steroids Supratarsal steroids Tacrolimus ointment	2% cyclosporine Oral steroids Cryotherapy Plaque debridement

6. Compare and contrast VKC and atopic keratoconjunctivitis (AKC). (5 marks)

Similarities

- Both VKC and AKC are severe and chronic types of allergic conjunctivitis
- They are treated on similar lines

Defferences

Feature	Vernal keratoconjunctivitis	Atopic keratoconjunctivitis
Age group affected	2nd decade Burn out by adulthood	Adults, 30–50 years Tends to persist till old age
Presentation	More in summer	Unrelated to seasonal variation
Associated skin allergy	None	Yes

Contd.

Feature	Vernal keratoconjunctivitis	Atopic keratoconjunctivitis
Cicatricial lid abnormalities	None	More common due to chronicity of the condition
Tarsal involvement	Upper	Lower
Papillae	Cobblestone papillae	Micropapillae
Corneal scarring and manifestations	Less common	More common
Association of cataract	None	Yes
Tear levels of IL-4	Low	High

7. Summarize phylctenular keratoconjunctivitis. (5 marks)

Phlyctenular Keratoconjunctivitis

A type IV hypersensitivity due to an endogenous or microbial protein (thought to be tubercular or staphylococcal protein).

Clinical Features

Symptoms

- Foreign body sensation
- Localized redness
- Lacrimation

Signs

- Simple phlyctenular conjunctivitis: A localized area of redness on either of the sides of the cornea, with a central avascular pinkish-white nodule
- Necrotizing phlyctenular conjunctivitis: Ulcer forms at the apex leaving a raw area
- Military phlyctenular conjunctivitis: Multiple ulcers arranged haphazardly

Corneal Association

- Sacrfulous ulcer: Due to ulceration of a limbal nodule
- Fascicular ulcer: Ulcer on the cornea with a leash of feeder vessels coming from the conjunctiva

8. Describe the clinical features of ocular cicatricial pemphigoid and its management. (5 marks)

Ocular Cicatricial Pemphigoid

A type II hypersensitivity reaction to the basement membrane of the conjunctiva and other mucosae.

Clinical Features

It is usually seen in individuals above the age of 60 years, and more in women.

Symptoms

- Patients present with insidious onset of bilateral ocular redness, foreign body sensation, and photophobia
- The disease is often misdiagnosed as dry eye and thus tends to have a chronic, untreated course

Signs

- The disease can have 4 stages:

Stage 1:	Chronic conjunctivitis, dry eye, and subepithelial fibrosis
Stage 2:	Forniceal shortening
Stage 3:	Symblepharon formation
Stage 4:	Surface kertinization

- In the inflammatory stage, there appear subconjunctival vesicles that tend to ulcerate
- Corneal manifestations include erosions, keratitis, perforation
- Lid signs include entropion and trichiasis

Management

- Topical lubricants, antibiotics, and steroids
- Systemic therapy includes the use of immunosuppressants
  - Mild disease: Dapsone
  - Moderate disease: Methotrexate
  - Severe disease: Intravenous methylprednisolone with cyclophosphamide
- Surgical intervention is required for severe dry eye and limbal stem deficiency
  - Punctal occlusion
  - Trichiasis and entropion surgery
- Ocular surface reconstruction

SHORT ANSWERS

1. Demonstrate the utility of the conjunctival allergy test. (3 marks)

- Conjunctival allergy tests are similar to skin allergy tests performed by a pulmonologist
- They are indicated in chronic allergic conditions like perineal allergic conjunctivitis, atopic conjunctivitis, vernal keratoconjunctivitis
- It is best performed by an ophthalmologist and a set of known allergens is placed in the form of topical drops
- The patient has to stop all medicines before the test and the allergen is administered as drops
- After 15–20 minutes, there is a reaction in the form of redness, chemosis, itching, and tearing in case of a positive response

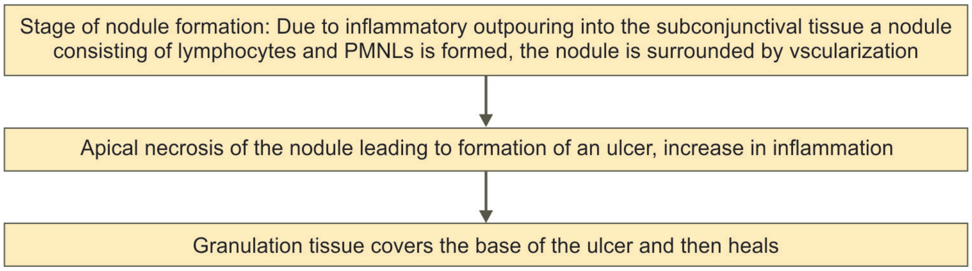
2. Describe eyelid signs associated with chronic conjunctival allergy. (3 marks)

Tylosis	Thickening of the tarsal border of the lids
Madarosis	Loss of eyelashes from the lids
Dennie–Morgan fold	The extralid folds due to chronic rubbing
Hertoghe's sign	Loss of lateral part of the eyebrows
Others	Trichiasis, entropion, ectropion

3. Explain the etiopathogenesis of phlyctenular conjunctivitis. (3 marks)

- Phlyctenular conjunctivitis is a type IV hypersensitivity due to an endogenous or microbial protein

- Commonly implicated organisms are *Mycobacterium tuberculosis*, *Moraxella axenfeld*, and staphylococci
- Systemic worm infestation is also another association
- However, in clinical practice, a source cannot be always found
- It is usually seen in girls, in developing countries, in poor nourished school-going children
- As a result of inflammation due to the abovementioned proteins, the following pathogenesis is proposed


Stage of nodule formation: Due to inflammatory outpouring into the subconjunctival tissue a nodule consisting of lymphocytes and PMNLs is formed, the nodule is surrounded by vscularization

Apical necrosis of the nodule leading to formation of an ulcer, increase in inflammation

Granulation tissue covers the base of the ulcer and then heals

4. Describe the causes and clinical features of giant papillary conjunctivitis. (3 marks)

Giant Papillary Conjunctivitis

- It was initially thought to be associated with contact lens wear
- It is also recognized with the presence of suture ends, prostheses, or filtering blebs

Causes

1. Use of prolonged contact lenses
2. The suture ends irritate the conjunctiva
3. Filtering blebs
4. Floppy eyelid syndrome

Symptoms

- Foreign body sensation is more pronounced than itching.
- Redness

Signs

- A hypermobile contact lens may be seen, in contact with the upper lid
- Suture ends may be seen
- Increased mucus production, protein-coated contact lens
- Macropapillae and giant papillae are arranged like cobblestone pavement
- There may be associated upper corneal erosions

5. Grade papillae based on size. (3 marks)

1. Micropapillae: Size <0.3 mm
2. Macropaillae: Size 0.3–1 mm
3. Giant papillae: Size >1 mm

COMPETENCY

OP3.6 DESCRIBE THE AETIOLOGY, PATHOPHYSIOLOGY, OCULAR FEATURES, DIFFERENTIAL DIAGNOSIS, COMPLICATIONS AND MANAGEMENT OF PTERYGIUM

LONG ESSAY

1. A 45-year-old farmer complains of a painless, fleshy mass noticed in the white of his eye noticed for 6 months. He has no visual symptoms.

A. What is the most probable diagnosis? (1 mark)
B. Explain the proposed etiopathogenesis of this condition. (3 marks)
C. Describe its clinical features and management. (5 marks)
D. Give two differential diagnoses. (1 mark)

A. Most Probable Diagnosis

Pterygium

B. Etiopathogenesis

- Pterygium is commonly seen in dry and sunny locations, and more commonly seen in men. Dry eye predisposes to the formation of pterygium
- The proposed etiopathogenesis is actinic (sunlight-induced) loss of limbal stem cells associated with elastotic degeneration and proliferation of the subconjunctival tissue. However, sunlight is not the only cause
- One of the important properties of the limbal stem cells is barrier function and it is lost due to the formation of pterygium
- Pterygium is more on the exposed part of the conjunctiva, thus supporting the above proposition. Also, it is more on the nasal side due to the reflection of the rays of sunlight off the nose onto the conjunctiva
- This tissue proliferates as a vascular, fleshy mass from the bulbar part of the conjunctiva onto the limbus and then on the cornea
- It invades the superficial stroma and the Bowman's membrane

C. Clinical Features and Management

Clinical Features

Symptoms

- Usually asymptomatic other than a fleshy mass seen in the eye
- Can cause foreign body sensation
- It induces against the rule astigmatism and hence can lead to blurred vision
- Once the pterygium encroaches the pupillary area, vision is disturbed

Signs

- Fleshy vascular mass
- There can be seen, a brown line, due to the deposition of iron at the head of the pterygium known as Stocker's line

Types (Dorothy Reed classification)

1. *Progressive:* It is usually highly vascular, fleshy and infiltration is seen ahead of the head
2. *Stationary:* It is pale, avascular, and thin, and lacks any infiltration

Management

- Only progressive pterygia need to be surgically excised
- Surgery: It involves the excision of the pterygium
    - Under aseptic conditions and local infiltration of anesthetic, a universal speculum is placed
- The body is then dissected under the conjunctiva and excised with Wescott's knife
- The bare area of the sclera is cauterized
- Options available after this stage are:
    - Leave the sclera bare (not practiced now)
    - Take a conjunctival graft from the superior or inferior conjunctiva of the same eye or fellow eye, place it on the bare sclera and suture it to the neighboring conjunctiva (low recurrence rate)
    - Use glue to stick the conjunctival autograft to the bare sclera
    - Can apply mitomycin C or thiotepa or beta irradiation
    - Amniotic membrane graft
- Postoperative antibiotics and steroids are given to decrease inflammation

D. Differential Diagnosis

1. Pseudopterygium
2. Ocular surface squamous neoplasia

SHORT ESSAY

1. Compare and contrast pterygium and ocular surface squamous neoplasms. (5 marks)

Pterygium and Ocular Surface Neoplasms

Feature	Pterygium	Ocular surface neoplasm
Type	Degenerative condition	Malignant condition
Pathology	Shows elastotic degeneration of the collagen with a fibrovascular proliferation of the subconjunctival tissue	Shows the presence of keratin pearls, hyperchromasia, irregular nuclear outline, and prominent nuclei There is a breach in the adenoid layer and Bowman's membrane of the conjunctiva.
Surface	Smooth	Usually uneven
Invasion	Invades superficial layers of the cornea and is free from underlying structures	Invades deep layers of the cornea and is fixed to underlying structures
Keratinization	Never keratinizes	May have keratinized surface

SHORT ANSWERS

1. Describe the method of using this drug in preventing the recurrence of pterygium. (3 marks)

- Mitomycin C is an antimetabolite that is used to decrease recurrences in pterygium surgery

- It is applied intraoperatively using a sponge in the concentration of 0.4 mg/ml and applied for 2–4 minutes below the cut edges of the conjunctiva
- It can also used as topical drops 0.2–0.4 mg/ml four times a day for 14 days

10 mg (potency)
℞ Mitomycin-C for Injection
Mitomycin-C Kyowa
10 mg.
Biochem

2. Describe various methods of decreasing recurrences following pterygium excision. (3 marks)

When the pterygium is excised and the sclera is left bare, the chance of recurrence is very high. So, to reduce the recurrence, one of the following methods may be adopted

1. **Conjunctival autograft**
    - A conjunctival graft is harvested from the superior or inferior bulbar conjunctiva of the same eye or fellow eye and placed on the bare sclera
    - It is sutured to the neighboring conjunctiva (low recurrence rate)
    - The same method is followed by the use of tissue adhesive to stick the conjunctival autograft to the bare sclera
2. **Use of antimetabolites**
    - Mitomycin C or thiotepa or beta irradiation
    - Mitomycin C inhibits DNA synthesis
    - It is applied for 3–5 minutes in a concentration of 0.02% to the bare sclera after excision of the pterygium
    - The bare sclera can be irradiated with thiotepa
3. **An amniotic membrane graft**
    - Applied to the bare sclera
    - It acts as a barrier to the growth of the pterygium

3. Differentiate between pterygium and pseudopterygium. (3 marks)

Pterygium and Pseudopterygium

Pterygium	Pseudopterygium
Elastotic degeneration leading to subconjunctival tissue growth and encroachment onto the cornea	It is a fold of conjunctiva due to inflammatory process which may be adherent to peripheral corneal ulcer
Always in palpebral aperture	Can be seen anywhere in the conjunctiva
Usually seen in elderly	Any age group
Can progress	Does not progress
Cannot pass a probe from beneath as it is subconjunctival tissue	Can pass a probe from beneath the tissue

4. Draw a neat labeled diagram of a pterygium mentioning its parts. (3 marks)

Parts of a Pterygium (Fig 3.6.1)

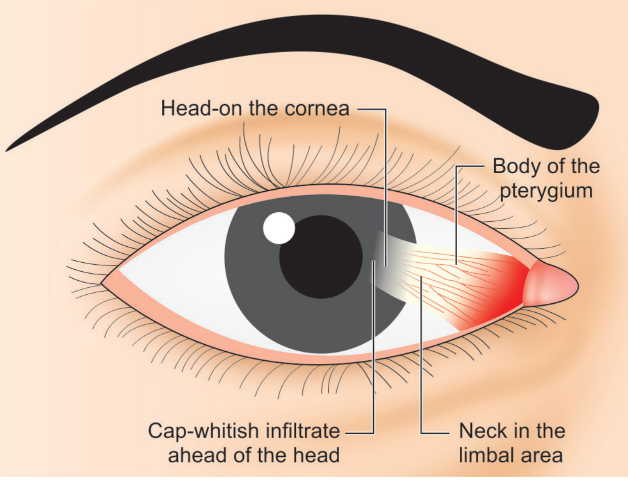


Fig. 3.6.1: Parts of a pterygium

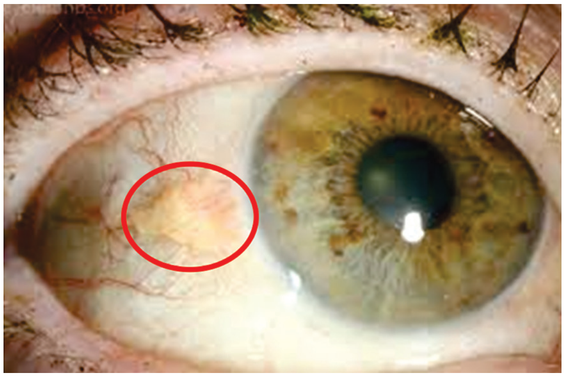
5. Explain the properties of a progressive pterygium vis-a-vis stationary pterygium. (3 marks)

Progressive and Stationary Pterygium

Feature	Progressive Pterygium	Stationary Pterygium
Appearance	Fleshy, vascular	Thin, atrophic, relatively avascular
Progression	Fast	Rare
Cap of infiltration	Present ahead of the head	Absent
Treatment	Surgical excision	Not required

6. Enumerate three conjunctival degenerations. (3 marks)

1. Pterygium
2. Pinguecula
3. Concretions



7. Identify the lesion shown in the image. Explain its management. (1+2 marks)

Lesion

Pinguecula

Management

- It usually does not need treatment as it does not give rise to any symptoms
- In case of ocular irritation, topical tear film substitutes can be administered
- Sometimes the pinguecula gets inflamed. These cases may be treated with a short course of topical steroids
- Simple excision is performed in cosmetic indications

COMPETENCY

OP3.7 DESCRIBE THE AETIOLOGY, PATHOPHYSIOLOGY, OCULAR FEATURES, DIFFERENTIAL DIAGNOSIS, COMPLICATIONS AND MANAGEMENT OF SYMBLEPHARON

SHORT ESSAY

1. A 26-year-old woman presents with Stevens–Johnson syndrome with ocular manifestations.

A. Enumerate the measures to be taken in the acute phase to prevent symblepharon? (3 marks)

B. Explain the definite management once symblepharon develops. (2 marks)

A. Measures to be taken at the acute presentation of Stevens–Johnson Syndrome (SJS)

- SJS is a hypersensitivity disorder characterized by skin and mucosal erosions
- The conjunctiva is frequently involved
- Systemic steroids are initiated
- Ocular care includes copious use of topical steroids to decrease the ongoing inflammation, tear film substitutes, and antibiotics
- If there are associated corneal erosions, patching the eye with ointment or bandage contact lens can be used
- A symblepharon shell or a scleral contact lens may be placed to keep the bulbar and the palpebral conjunctiva separate
- Forniceal sweeping is done twice a day to break if any symblepharon has developed It is done under topical anesthesia with a glass rod coated with a lubricating ointment

B. Management of Symblepharon

- It is indicated if the symblepharon is causing complications
- Surgery for symblepharon involves removal of the cicatrix and application of mitomycin C
- It is followed by tissue grafting to the affected areas and reconstruction of a normal fornix
- Tissue grafts can be harvested from oral mucosal transplantation, conjunctival autografting, or amniotic membrane transplantation
- Cultivated limbal stem cell transplantation is another surgical procedure to treat symblepharon that can be used in cases of severe burns

SHORT ANSWERS

1. Define a symblepharon? Enumerate two causes for the same. (1+2 marks)

Definition of Symblepharon

An adhesion between the bulbar and the palpebral conjunctiva.

Causes

1. Chemical injury
2. Stevens–Johnson syndrome

2. Define ankyloblepharon. Enumerate two causes for the same. (1+2 marks)

Definition of Ankyloblepharon

An adhesion between the upper and lower eyelids.

Causes

1. It may be congenital or acquired
2. Causes for acquired ankyloblepharon: Chemical injury, Stevens–Johnson syndrome

3. Describe the clinical manifestations and implications of a symblepharon. (1+2 marks)

Symblepharon

Clinical Manifestations

- In mild cases, it may go unnoticed
- In severe cases, there may be restricted eye movements or eye closure

Implications

- Symblepharon may incidentally be discovered and signifies an old ocular surface inflammation
- It may be associated with mucin deficiency dry eye as the etiology of both are similar
- Restricted eye movements may be confused with nerve/muscle palsies
- Decreased lid movement may be confused with ptosis or lagophthalmos and thus conjunctival and corneal xerosis, thickening, and keratinization may occur due to exposure

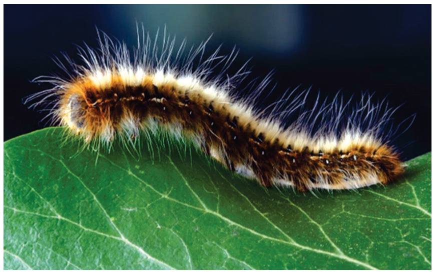
COMPETENCY

OP3.8 DEMONSTRATE CORRECT TECHNIQUE OF REMOVAL OF FOREIGN BODY FROM THE EYE IN A SIMULATED ENVIRONMENT

SHORT ESSAYS

1. State the ocular condition caused by the hair of this organism. Explain the clinical features and its management. (1+2+2 marks)

Ocular Condition Caused by the Hair of Given Organism



- Ophthalmia nodosa
- It is a clinical condition characterized by an allergic reaction of the ocular surface to caterpillar hair
- The caterpillar hair is extremely sharp and tends to penetrate the cornea
- They also mount severe allergic reactions

Ophthalmia Nodosa

Clinical Features

- There can be severe follicular hypertrophy when the hair is embedded in the conjunctiva
- In the conjunctiva, a nodule on the bulbar conjunctiva can be seen
- Corneal erosion due to rubbing can be seen
- Sometimes, the hair is seen to be entering the cornea to various depths
- It can incite uveitis if enters the anterior chamber

Management

- Removal of caterpillar hair under the view of a slit-lamp under topical anesthesia
- Use of topical steroids to decrease inflammation
- Use of tear film substitutes to promote healing of corneal erosions
- The nodule needs to be surgically excised under the cover of steroids

2. Name four common conjunctival foreign bodies. Describe their clinical features and implications. (1+2+2 marks)

Common Foreign Bodies

- Metal while grinding (industrial)
- Sand or dust while driving
- Insect/or its part
- Wood while sawing

Clinical Features

- There is always a history of sudden onset of irritation and foreign body sensation
- Redness and watering
- In presence of the upper palpebral conjunctival foreign body, it may cause corneal erosions
- The pattern of corneal erosions may help to localize the foreign body

Implications

- If it is superficial, it causes localized infection in the conjunctiva
- If it is deep, it can cause preseptal cellulitis with lymphadenopathy
- They can be granuloma formation around the foreign body

SHORT ANSWERS

1. Explain in brief the method of removing a conjunctival foreign body. (3 marks)

- The foreign body is localized by a careful slit-lamp examination
- Topical proparacaine is instilled
- Particulate matter can be removed with a cotton-tipped applicator after instillation of topical anesthesia (proparacaine)
- Foreign bodies embedded deep in the tissue need to be removed with a hypodermic needle (26 gauge, half an inch)
- A Jeweler's forceps, blunt spatula, or epilation forceps (blunt ends) can be used to pick the foreign body
- This is followed by the instillation of a topical antibiotic

2. Explain the method of ocular surface irrigation. Give two indications. (2+1 marks)

Method of Ocular Surface Irrigation

- The patient is made to lie down in a supine position
- Topical proparacaine is instilled
- An infusion system is set in place with the irrigation cannula in the examiner's right hand
- The left hand is used to evert the lids
- A kidney tray is placed near the ear of the patient and the head is tilted in the same direction
- The ocular surface is irrigated with normal saline by everting the upper and lower lids alternatively
- A Desmarre's retractor is used if the foreign body cannot be localized

Indications

1. Chemical injury
2. Superficial conjunctival foreign body

3. Appraise the importance of sulcus subtarsalis. (3 marks)

- Sulcus subtarsalis is a narrow physiological groove that is seen 2 mm posterior to the lid margin
- It marks the transition between skin and conjunctiva
- It is a frequent site for lodgment of foreign bodies
- Foreign bodies in sulcus subtarsalis may cause corneal erosions, as they are close to the cornea
- In case of multiple minute foreign bodies, ocular surface irrigation is done

4. Explain eversion and double eversion. (3 marks)

Eversion and double eversion are methods to examine the palpebral conjunctiva and fornices.

Eversion

- The lower lid is easy to evert
- The skin is pulled down with the tip of the thumb to expose the lower palpebral conjunctival and the lower fornix
- The upper lid is everted by using a cotton bud
- It is placed at 4–5 mm from the lid margin and the lashes are used to gently pull the lid away from the globe.
- The lid is then rolled on itself

Double Eversion

- It is done using a Desmarre's retractor and is useful for the examination of the upper fornix
- The Desmarre's retractor is placed on the upper lid with its concavity anteriorly and the lashes are held gently and the lid margin is 'fed' into the concavity of the retractor
- Now, the retractor is turned upwards to reveal the superior fornix

COMPETENCY

OP3.9 DEMONSTRATE THE CORRECT TECHNIQUE OF INSTILLATION OF EYE DROPS IN A SIMULATED ENVIRONMENT

SHORT ESSAYS

1. Describe the methods of administration of drugs onto the ocular surface. (5 marks)

Drugs can be administered to the eye in various methods of which topical is the most commonly employed method

1. **Topical eye drops as solutions and suspensions**
    - Drugs are formulated into solutions and suspensions of various concentrations
    - They are instilled in the conjunctival cul-de-sac by gently everting the lower lid and tilting the patient's head
    - Only one drop is instilled as more volume than 20 µl will overflow
    - Some drug vehicles like methylcellulose and polyvinyl alcohol increase corneal contact time

- Drugs like benzalkonium chloride (BKC) increase penetration by breaking the tear film
- Many drugs can be stored at room temperature, except for a few antiglaucoma drugs that need to be refrigerated
- *Advantages:* Drug is available for immediate action
- *Disadvantage:* It gets drained into the nasolacrimal system and gets absorbed systemically

2. **Topical ointments**
   - These are best suited for bedtime application
   - Commonly used drugs are atropine (for cycloplegia in children), antibiotics, steroids, and so on
   - *Advantage:* Prolonged contact time, no systemic absorption
   - *Disadvantage:* Interferes with vision, uncomfortable
3. **Inserts or ocuserts**
   - These are small devices containing drugs and the drug diffuses into the conjunctival space through a membrane
   - It is placed in the lower or upper fornix for about a week. Pilocarpine is an example
   - *Advantage:* There is a slow delivery of the drug so patient compliance is better
   - *Disadvantage:* There is a rare possibility of damage to the ocusert and potential toxicity
4. **Iontophoresis**
   - Here the drug is delivered by using a small volt of galvanic current
   - It is rarely used
5. **Hydrophilic contact lens**
   - Hydrogel bandage contact lenses are soaked in the required drug overnight and applied over the cornea
   - The drug is slowly released into the conjunctival sac

2. Describe the factors that influence the absorption of the drug from the ocular surface. (5 marks)

- When a drug is topically instilled, then it mainly gets absorbed through the cornea
- Hydrophilic drugs are mainly absorbed through the conjunctival and scleral vessels
- They finally reach the aqueous humor
- The drug has to overcome two important barriers: Tear film and the corneal epithelium

1. **Factors that help overcome tear film**
   - Some agents added to the drug helps in breaking the tear film
   - An example is BKC, a common drug preservative. It has a detergent-like action on the tear film and helps in greater penetration
2. **Nature of the drug**
   - Lipophilic or hydrophobic drugs easily penetrate the epithelium as it is lipophilic
   - The stroma is hydrophilic, so drugs with hydrophilic properties are absorbed from the stroma
   - So ideally, a drug should have both polar and nonpolar properties
   - The size of the molecules also affects absorption. The smaller the drug, the easier is the absorption
   - Increased viscosity of the drug causes an increase in contact time with the cornea
   - The pH of the drug should be neutral. Increased or decreased pH causes ocular irritation and reflex lacrimation that causes increased drainage

3. **Nasolacrimal drainage**
    - About 80% of the drug instilled gets drained via the nasolacrimal system
    - It is increased by increased blinking
    - When the patients are refrained from blinking, the drug evaporates from the ocular surface
    - So, it is ideal to close the lids after instillation of the drug with punctal occlusion

SHORT ESSAYS (MISCELLANEOUS)

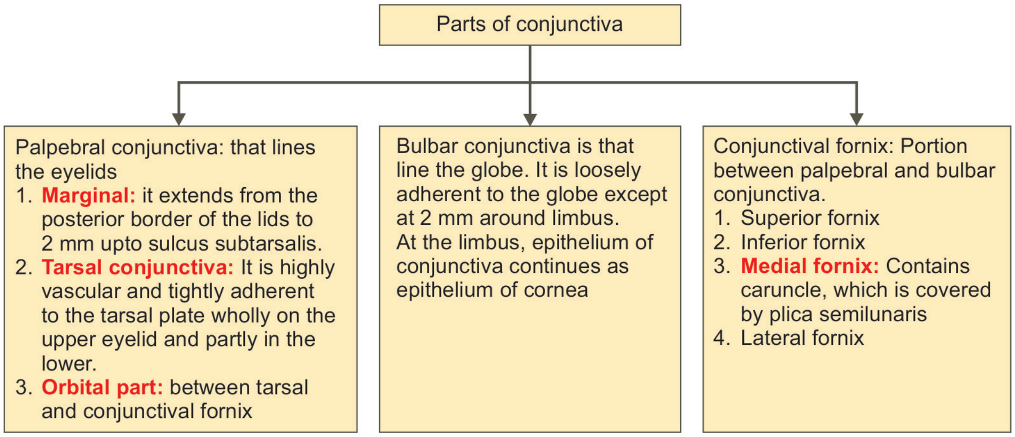
1. Describe the uses of fluorescein staining in the eye. (5 marks)

- Fluorescein is an organic, hydrophilic dye that is frequently used in ophthalmology
- It is excited by blue light (430 nm) and emits green light (525 nm)
- It is mainly used for ocular surface disorders and angiography
- A blue filter is used to excite the dye and the dye is recognized by green light

1. **Uses in the ocular surface**
    - Epithelial defects on the ocular surface stand out green when seen under blue light
    - It is commonly used to rule out and diagnose corneal epithelial loss in abrasions, ulcers, superficial punctate keratopathies, UV keratopathy, and chemical injuries
    - It also detects defects in the epithelium of the conjunctiva in chemical injuries, trauma, and conjunctival ulcers
    - When fluorescein is instilled in the conjunctival sac, it stains the dried epithelium and is suggestive of dry eyes
    - It is used to diagnose a woud leak or corneal perforation by Siedel's test
    - Fluorescein dye disappearance test and Jones test are used in the diagnosis of epiphora.
        - In this test, fluorescein dye is instilled in the conjunctival sac and the time taken for it to disappear from the ocular surface is noted.
        - A period of more than 5 minutes is diagnostic of epiphora or incomplete drainage
    - Tear film break-up time
        - After instilling fluorescein, the patient is asked to blink and then keep eye open
        - The eye is examined under blue light and the time taken for the first dry patch to appear on the cornea is noted
        - Normally, it is around 15–30 seconds. Lesser than 10 seconds is suggestive of dry eye
        - It detects all types of dry eyes
2. **Applanation tonometry**
    - Goldman applanation tonometry is the gold standard for measuring intraocular pressure
    - In this test, the cornea is anesthetized by using proparacaine and fluorescein ins instilled
    - When the prisms meet the cornea, green rings are formed. The knob is turned to approximate the rings to give the value of IOP
3. **Fluorescein angiography**
    - In this test, fluorescein is injected into the antecubital vein and serial fundus photographs are taken
    - This test helps to study various pathologies of the retinal and sometimes choroidal vasculature

2. Describe various parts of conjunctiva and add a note on its microscopic layers. (3 + 2 marks)

Parts of Conjunctiva



Parts of conjunctiva

Palpebral conjunctiva: that lines the eyelids

1. **Marginal:** it extends from the posterior border of the lids to 2 mm upto sulcus subtarsalis.
2. **Tarsal conjunctiva:** It is highly vascular and tightly adherent to the tarsal plate wholly on the upper eyelid and partly in the lower.
3. **Orbital part:** between tarsal and conjunctival fornix

Bulbar conjunctiva is that line the globe. It is loosely adherent to the globe except at 2 mm around limbus. At the limbus, epithelium of conjunctiva continues as epithelium of cornea

Conjunctival fornix: Portion between palpebral and bulbar conjunctiva.

1. Superior fornix
2. Inferior fornix
3. **Medial fornix:** Contains caruncle, which is covered by plica semilunaris
4. Lateral fornix

Histology

- Conjunctiva has three layers: Epithelium, adenoid layer, and fibrous layer (Fig. 3.M.1)
- Epithelium varies from region to region
  - The epithelium is stratified squamous nonkeratinized
  - The marginal epithelium is made of 5 layers of stratified squamous epithelium
  - Tarsal conjunctiva has an external layer of cylindrical cells and inner flat cells
  - Fornix and bulbar conjunctiva have a superficial layer of cylindrical cells, a middle layer of polyhedral cells, and a deep layer of cuboidal cells
  - Limbal conjunctiva has 5–6 layers of tightly packed stratified squamous epithelium
- Adenoid layer: It consists of lymphocytes in connective tissue.
- Fibrous layer: It consists of a meshwork of collagenous and elastic fibers. This layer carries blood vessels and nerves of the conjunctiva

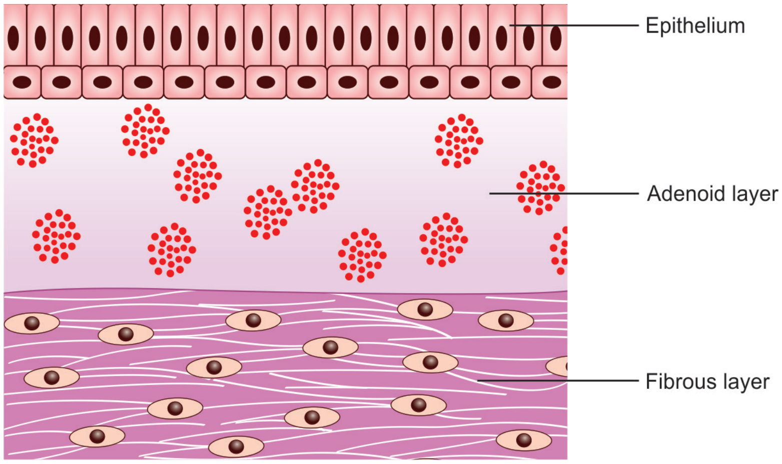


Fig. 3.M.1: Histology of conjunctiva

3. Describe the nerve supply and lymphatic drainage of the conjunctiva and its clinical implications. (5 marks)

Nerve Supply

- Limbal conjunctiva is supplied by the ophthalmic division of the trigeminal nerve
- Rest is supplied by lacrimal, infratrochlear, supratrochlear, supraorbital, and frontal nerves

Lymphatic Drainage

- Lateral two-thirds of the upper palpebral conjunctiva and lateral one-third of the lower palpebral conjunctiva drain into the preauricular nodes
- Medial one-third of the upper palpebral conjunctiva and medial two-thirds of the lower palpebral conjunctiva drain into the submandibular lymph nodes

Clinical Implications

- Preauricular lymph nodes are inflamed in many types of viral conjunctivitis, chlamydial conjunctivitis
- Ocular surface squamous neoplasia is associated with preauricular and submandibular lymphadenopathy
- Conjunctival sensations are responsible for the identification of foreign bodies
- Conjunctival sensations are lost in trigeminal nerve pathologies. They are tested clinically using a wisp of cotton

SHORT ANSWERS

1. Describe the appearance of Bitot spots. Give two causes. (2+1 marks)

Bitot Spots

- These are dry areas on the bulbar conjunctiva, usually seen on either side of the cornea
- Classically seen in association with vitamin A deficiency, due to goblet cell deficiency, the conjunctiva becomes dry and appears brown
- A bacterium, *Corynebacterium xerosis*, thrives well in this environment and produces foam-like secretion which gives this silvery appearance to the Bitot spots

Causes

1. Vitamin A deficiency
2. Ocular cicatricial pemphigoid

2. Describe the clinical features of conjunctival melanoma. (3 marks)

Malignant melanoma may arise due to malignant transformation of an existing nevus or arise *de novo*.

Symptoms

Patients may notice a sudden increase in the size of a preexisting nevus.

Signs

- It may be seen as a pigmented, vascular mass near the limbus, usually fixed to the epibular tissue
- There may be associated lymphadenopathy
- It does not penetrate the globe, but distant metastases are well known

CHAPTER

4

Corneas

COMPETENCY

OP4.1 ENUMERATE, DESCRIBE AND DISCUSS THE TYPES AND CAUSES OF CORNEAL ULCERATION

LONG ESSAYS

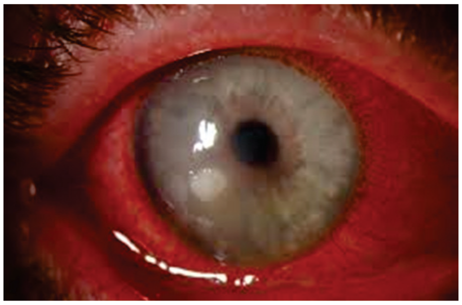
1. A 35-year-old diabetic presents with severe ocular pain, redness, and diminution of vision. On examination, the following image is seen.

A. State the most probable diagnosis? (1 mark)

B. Which organism has probably caused this? (1 mark)

C. Explain its stages and natural history. (4 marks)

D. Illustrate the color-coding of the anterior segment drawing and documentation. (4 marks)



A. Most Probable Diagnosis

Corneal ulcer

B. Organism Responsible

The most probable cause is gram-positive organisms like *Staphylococcus* and *Streptococcus*

C. Stages of Corneal Ulcer and Their Natural History

1. **Stage of infiltration (Fig. 4.1.1)**
   - Following entry of pathogens, there is characterized by the infiltration of polymorphonuclear and/or lymphocytes into the epithelium from the limbal vessels and underlying stroma
   - An inflammatory response ensues

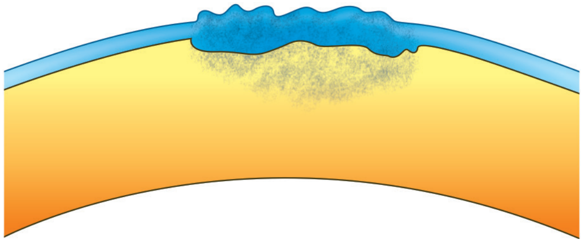


Fig. 4.1.1: Stage of infiltration

2. **Stage of active ulceration (Fig. 4.1.2)**
    - Depending on the virulence of the organism and immunity of the host, the inflammation brought about causes necrosis of the tissue
    - This is a protective mechanism to destroy pathogens
    - As a result, there increase in the size of the ulcer once the host immune system starts its function
    - An immune ring is seen around the ulcer
    - The unhealthy epithelium sloughs off. The edges and base of the ulcer are edematous and show grayish white exudates
    - There is ciliary congestion, anterior chamber reaction, and hypopyon formation

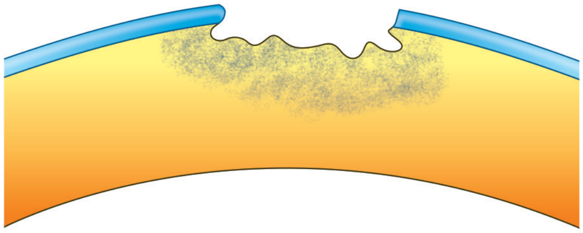


Fig. 4.1.2: Stage of active ulceration

3. **Stage of healing (Fig. 4.1.3)**
    - In this stage, either due to treatment or host immune response, the pathogens are eliminated
    - The reparative process is marked by epithelial regrowth and laying down of collagen by keratocytes.
    - An immune ring is seen around the ulcer
    - The corneal clouding decreases and the ulcer starts healing

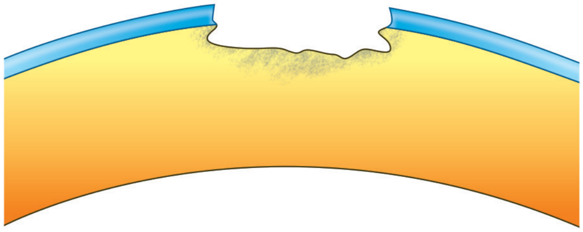


Fig. 4.1.3: Stage of healing

4. **Stage of cicatrization (Fig. 4.1.4)**
    - In this stage, collagen fibers are laid down irregularly and as a result, the clarity of the cornea is compromised
    - As a result, an opacity forms
    - Peripheral corneal ulcers get vascularized

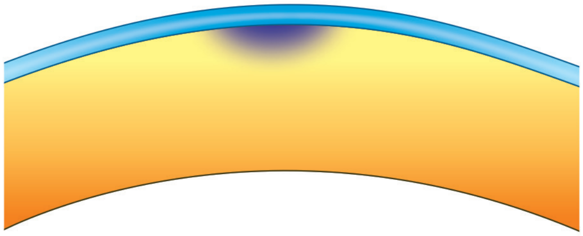


Fig. 4.1.4: Stage of cicatrization

D. Color-coding of the Anterior Segment (Fig. 4.1.5)

1. **Red**
    - Blood vessels
    - Hyphema
    - Ghost vessels, vascularization of the cornea
2. **Blue**
    - Epithelial and stromal edema
    - Descemet's folds
3. **Black**
    - Scars
    - Dystrophies
    - Pigments

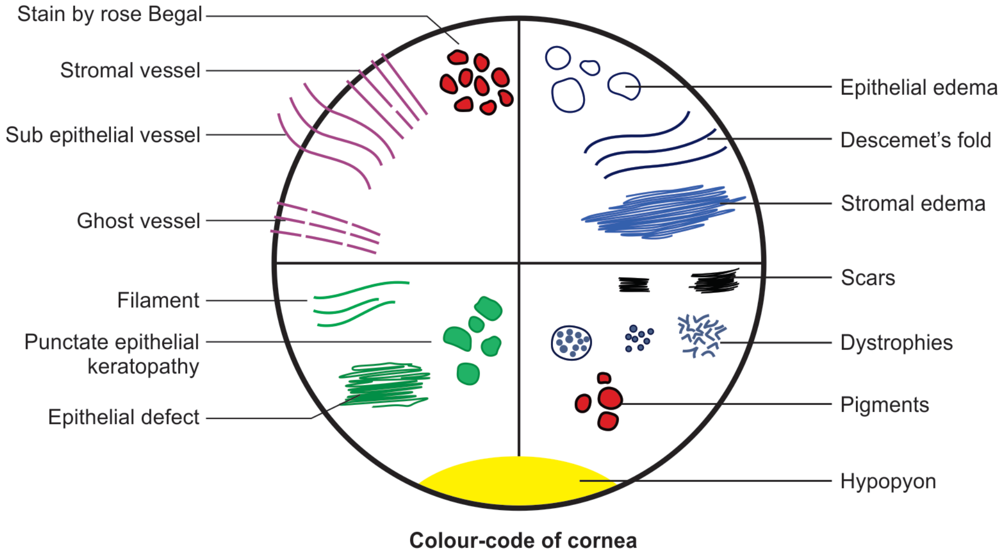

Stain by rose Begal
Stromal vessel
Sub epithelial vessel
Ghost vessel
Filament
Punctate epithelial keratopathy
Epithelial defect
Epithelial edema
Descemet's fold
Stromal edema
Scars
Dystrophies
Pigments
Hypopyon
Colour-code of cornea

Fig. 4.1.5: Color-coding of anterior segment drawing

4. **Green**
   - Epithelial defects
   - Epithelial erosions
5. **Yellow**
   - Slough
   - Hypopyon

2. A 60-year-old woman with a corneal ulcer complains of sudden pain in the affected part with a gush of water-like material from the eye.

A. State the most probable diagnosis? (1 mark)

B. Enumerate various complications of corneal ulcers. (3 marks)

C. Explain the management of imminent perforation and perforated corneal ulcer. (4 marks)

D. Explain the pathogenesis of anterior staphyloma. (2 marks)

A. Most Probable Diagnosis

Perforation of a corneal ulcer.

B. Complications of a Corneal Ulcer

- Toxic iridocyclitis
- Secondary glaucoma: Can occur due to associated inflammatory exudates clogging the trabecular meshwork
- Descemetocele and perforation: Sequelae of perforation
  1. Incarceration of iris into the ulcer
  2. Anterior lens subluxation and anterior capsular cataracts may occur due to sudden forward movement of the iris lens diaphragm
  3. Corneal fistula

4. Endophthalmitis and panophthalmitis can occur due to the entry of organisms into the vitreous
5. Intraocular hemorrhage in the form of either vitreous hemorrhage or expulsive choroidal hemorrhage may occur in some patients due to sudden lowering of intraocular pressure
6. Corneal opacity

C. Management of Imminent Perforation

- Treat problems that can increase intraocular pressure: give stool softener, antitussives if cough
- Decreasing IOP using oral acetazolamide
- Pressure bandage of the eye
- Bandage contact lens
- Tissue adhesive like cyanoacrylate

Management of Perforated Corneal Ulcer

- The perforated corneal ulcer should be treated with a tissue adhesive if the perforation <2 mm in size. Tissue adhesive like cyanoacrylate is placed over the perforation and a bandage contact lens is placed
- Tectonic keratoplasty is performed to restore the integrity of the globe

D. Pathogenesis of Anterior Staphyloma

- When a virulent organism like *Pseudomonas* and *Klebsiella* leads to spreading sloughing corneal ulcer, involving >2/3rds of the surface, exudates block the pupil and cover the iris tissue and form a pseudocornea
- As it cannot withstand intraocular pressure, it bulges forward.
- It resembles a bunch of grapes and hence is called an anterior staphyloma
- It has a high chance of bleeding

SHORT ESSAYS

1. A 33-year-old woman with erythematous lesions over the cheeks presents with a painless swelling in the eyelid.

A. Enumerate the manifestations of this skin condition in the cornea. (1 mark)

B. Explain the management of this condition (corneal manifestations). (2 marks)

C. Give two examples of drug-induced keratopathy. (2 marks)

A. Manifestations of this Skin Condition

- The dermatological condition explained in this scenario is acne rosacea
- The ocular manifestations are:
  - Dry eye
  - Superficial punctate erosions of the cornea
  - Corneal vascularization
  - Marginal corneal ulcer and perforation

B. Management of Marginal Corneal Ulcer

- Topical steroids/cyclosporine along with cycloplegics and lubricants for the ulcer
- Systemic therapy in the form of oral tetracycline 250 mg QID for 3 weeks, TDS for 3 weeks, BID for 3 weeks, and once a day for 3 months
- Other options are doxycycline and oxytetracycline

C. Examples of Drug-induced Keratopathy

1. Vortex keratopathy is caused by Amiodarone, Hydroxychloroquine, Chlorpromazine, Tamoxifen, and so on
2. Crystalline punctate deposits after topical Ciprofloxacin administration
3. Punctate keratopathy with intraepithelial opacities with the use of Cytarabine

2. Draw a neat and labeled diagram of the layers of cornea and mention one special property of each. (2+3 marks)

Layers of Cornea (Fig. 4.1.6)

Special Properties of Corneal Layers

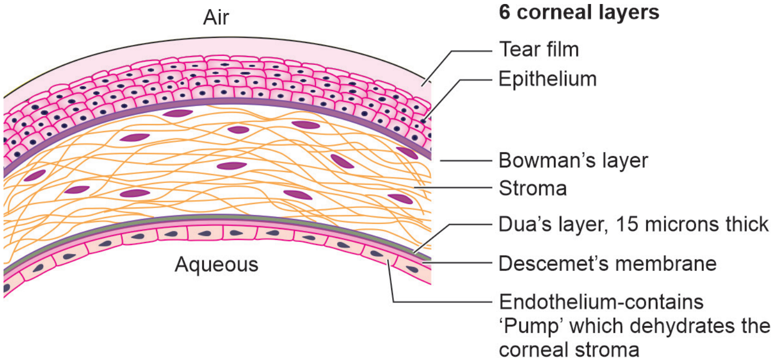

Air
6 corneal layers
Tear film
Epithelium
Bowman's layer
Stroma
Dua's layer, 15 microns thick
Descemet's membrane
Endothelium-contains 'Pump' which dehydrates the corneal stroma
Aqueous

Fig. 4.1.6: Layers of the cornea

1. **Epithelium**
    - It is a nonpolar layer
    - Consisting nonkeratinized stratified squamous epithelium of 5–6 layers, 2–3 umbrella-shaped cells, and a basal layer of columnar cells
2. **Bowman's membrane**
    - Anterior condensation of stroma made of collagen fibrils, forms barrier to infection, does not regenerate
3. **Stroma**
    - It forms 90% of the thickness of the cornea, is a hydrophilic part, consists of proteoglycans and collagen fibrils laid parallel to each other in several layers
    - The arrangement is so uniform that these fibrils are parallel to the corneal plane also
    - Among the lamellae are present keratocytes, wandering macrophages, histiocytes, and a few leucocytes
    - Keratocytes form collagen
4. **Dua's layer**
    - Newly discovered layer
    - 15 microns thick
5. **Descemet's membrane**
    - It forms the basement membrane of the corneal endothelium and is a strong homogeneous layer that is resistant to chemical agents, trauma, and pathological processes

- It is made up of collagen and glycoproteins. When torn, it curls around itself and can regenerate
- In the periphery, it appears to end at the anterior limit of trabecular meshwork as Schwalbe's line (ring)

6. **Endothelium**
   - It is a single layer of cells with cannot regenerate
   - They tend to decrease in number with age
   - They are normally hexagonal and are around 4000 cells/mm^3 at the age of 25 years. With the loss of endothelium due to age, disease process, or trauma, they glide over to cover the defect and thus acquire polymorphous shapes (polymegathism)
   - <600 cells/mm^3 lead to corneal edema due to entry of aqueous and hydration of stroma

3. Classify ulcerative keratitis based on etiology. (5 marks)

1. Infectious keratitis
   1. Bacterial
   2. Viral
   3. Fungal
   4. Chlamydial
   5. Protozoal
   6. Spirochaetal
2. Allergic keratitis
   1. Phlyctenular keratitis
   2. Vernal keratitis
   3. Atopic keratitis
3. Trophic keratitis
   1. Exposure keratitis
   2. Neuroparalytic keratitis
   3. Keratomalacia
   4. Atheromatous ulcer
4. Keratitis associated with diseases of the skin and mucous membrane
5. Keratitis associated with systemic collagen vascular disorders
6. Traumatic keratitis, which may be due to mechanical trauma, chemical trauma, thermal burns, radiations
7. Idiopathic keratitis, e.g., Mooren's corneal ulcer, superior limbic keratoconjunctivitis, Superficial punctate keratitis of Thygeson

4. Describe the clinical features of microscopic perforation (or microperforation) of a corneal ulcer. (5 marks)

- Frank perforations are easily recognized by a flat anterior chamber, iris prolapse, and an ulcer that shows perforation
- Subtle microscopic perforations can be missed if not looked for
- The patients may complain of on- and- off tearing from the eye
- It should also be suspected when the ulcer is not healing with medications
- On examination, one can see Descemet's folds radiating from the ulcer
- The center of the ulcer shows a descemetocele that may not be frankly leaking. A Siedel's test is performed by instilling a drop of fluorescein in the conjunctival sac and examining it under blue light. Dilution of the dye is seen as a greenish hue at the descemetocele. This is called 'filtering descemetocele'. This is classical of a microperforation

- Also, the anterior chamber is uneven or shallow. It may not necessarily be flat as in a frank perforation. The IOP is usually <10 mm Hg
- Another important feature of a microperforation is corneal hemorrhage

5. Elaborate 5 signs of corneal inflammation. (5 marks)

1. **Superficial punctate erosions**
   - Point-sized epithelial defects that stain with fluorescein and rose Bengal caused by a variety of causes
   - Their position and pattern will help diagnosis
     - Superior SPEs are seen in superior limbic keratoconjunctivitis, floppy eyelid, ill-fitting contact lenses
     - Inferior SPEs are seen in lagophthalmos, blepharitis, drug toxicity
     - Interpalpebral SPEs are seen in UV keratopathy, dry eyes, and reduced corneal sensations
2. **Punctate epithelial keratitis**
   - Granular swollen epithelial cells, stain poorly with fluorescein but well with rose Bengal stain
   - They are seen in association with adenoviral conjunctivitis as a host immune response, staphylococcal hypersensitivity, Thygeson superficial keratopathy, and chlamydial infections
3. **Filamentary keratopathy**
   - Filaments are strands of epithelium with one end stuck to its base and the other mobile with every blink
   - They stain with fluorescein
   - It is seen in severe dry eye, corneal epithelial instability, superior limbic keratopathy, and neurotrophic keratopathy
4. **Epithelial edema**
   - It is characterized by a loss of transparency and normal sheen of the cornea and is replaced with ground glass-like opacification
   - Edema is seen surrounding corneal inflammation
5. **Infiltrates**
   - It is seen in the stroma as a part of the immune response mounted by the cornea due to which there is a concentration of leukocytes and cellular debris
   - It is seen as grayish white irregular opacities in the superficial stroma associated with ciliary congestion
   - It is surrounded by less dense opacities
   - Can be sterile or purulent
   - Purulent infiltrates have these following features
     - P: Pain
     - E: larger epithelial defects (>1 mm)
     - D: Discharge
     - A: Anterior chamber reaction and hypopyon
     - L: more central location

6. Describe the clinical features of a corneal ulcer. (5 marks)

Corneal Ulcer

Clinical Features

Symptoms

- Pain and lacrimation
- Redness
- Foreign body sensation
- Decreased vision
- Photophobia

Signs

- Visual acuity is decreased on the location and severity of surrounding corneal edema
- Lid edema
- Ciliary congestion
- A corneal ulcer is seen as an epithelial defect with surrounding grayish white infiltrates. Surrounding corneal haze is seen. The floor of the ulcer is covered with exudates and margins are overhanging
  - Staphylococci usually cause well-demarcated deep ulcers with minimal corneal edema
  - *Pseudomonas* causes a superficial, sloughing ulcer with irregular margins and diffuse corneal edema
  - Enterobacteria causes a superficial ulcer with surrounding corneal edema and an immune ring due to endotoxins
- Anterior chamber reaction may be seen in some ulcers with hypopyon

7. Describe etiology, clinical features, and management of neurotrophic keratopathy. (1+2+2=5 marks)

Neurotrophic Keratopathy

Etiology

- Damage to the trigeminal nerve due to stroke, neoplasms, inflammation, or trauma
- Systemic diseases like diabetes and leprosy
- Ocular diseases like herpetic eye disease, local anesthetic abuse, incisional refractive surgeries
- Congenital disorders like Riley day syndrome, Goldenhar syndrome, hereditary sensory neuropathy, and anhidrotic ectodermal dysplasia

Clinical Features

- Patients present with minimal symptoms
- There are no symptoms like pain, lacrimation, and photophobia as there are no sensations
- The epithelium has an irregular appearance in the interpalpebral zone and shows punctate erosions
- An ulcer may form with rolled edges and loose surrounding epithelium
- There is associated stromal edema
- Corneal sensations are reduced

Management

- Lubricants such as polyvinyl alcohol, hypromellose, carboxymethyl cellulose
- Ocular padding and bandaging or even simple taping

- Bandage contact lens
- Tarsorrhaphy
- Amniotic membrane transplant over the nonhealing ulcer
- Topical insulin-like growth factor, substance P, and neurogenic factor are being investigated.

8. Describe the management of an uncomplicated corneal ulcer. (5 marks)

Management of an Uncomplicated Ulcer

Ulcer debridement and microbiological examination:

- The margins of the ulcer are gently debrided with a Kimura's spatula or a blade after instilling topical anesthesia
- The scrapings from the ulcer are sent for microbiological examination like Gram's stain, KOH mount (for fungi), and are inoculated into blood agar, chocolate agar, and SDA agar
- Rule out a blocked nasolacrimal duct by doing a lacrimal syringing
- Rule out diabetes as it is a common cause of a nonhealing ulcer
- Analgesics and anti-inflammatory drugs are given to increase patient comfort and decrease inflammation
- Cycloplegics are started right away to relax the ciliary muscle and decrease the pain. It brings down associated uveitis. The drugs that can be used are atropine, homatropine, and tropicamide
- Antibiotics are started after scrapings have been sent for microbiological examination.
- It can also be started based on presumptive diagnosis based on the characteristics of the ulcer. Later, based on sensitivity, they can be changed.

Gram-positive cover	Gram-negative cover
• Fluoroquinolones like ciprofloxacin, ofloxacin, Gatifloxacin, and moxifloxacin (0.3%) • Cephazolin 75–100 mg/ml • Aminoglycosides like gentamicin (14 mg/ml), tobramycin (14 mg/ml) • Vancomycin (33 mg/ml)	• Aminoglycosides like gentamicin (14 mg/ml), tobramycin (14 mg/ml)

- Secondary glaucoma is the most common cause of a nonhealing ulcer. It may be caused by intense inflammation as evidenced by the formation of a hypopyon. Hence, topical beta-blockers or carbonic anhydrase inhibitors are given to bring down IOP.

9. A 55-year-old man presents with a corneal ulcer. He is started on antimicrobials after laboratory diagnosis of the organism. However, there is no improvement in the ulcer even after one week of therapy. Enumerate three causes for this condition and describe their management. (2+3 marks)

This patient has a nonhealing corneal ulcer.

Cause	Management
High intraocular pressure	• It is usually caused by a hypopyon as it interferes with the drainage of the aqueous • As a result, the removal of toxic materials from the aqueous is delayed • The ulcer heals slowly • To manage this antiglaucoma medications like beta-blockers and carbonic anhydrase inhibitors are used

Contd.

Cause	Management
Blocked nasolacrimal duct	• A blocked nasolacrimal duct predisposes to chronic dacryocystitis • As a result, there is continuous availability of bacterial that keep getting collected in the tears • To treat this a dacryocystectomy should be performed
Uncontrolled diabetes	• Uncontrolled diabetes and resultant hyperglycemia are a cause for any kind of infection flare-up in the body • It also contributes to sensory neuropathy of the cornea and delays healing by decreasing the trophic factors • Control of blood sugars with lifestyle modification and using suitable medications (oral hypoglycemic agents and insulin) should be done

10. Describe the clinical features and management of peripheral ulcerative keratitis. (2+3 marks)

Peripheral Ulcerative Keratitis

Clinical Features

- Initial symptoms are quite nonspecific and include foreign body sensation, lacrimation, and photophobia
- The examination may reveal epithelial erosions and ulceration in the peripheral cornea adjoining the sclera
- This may induce astigmatism
- There may be associated anterior uveitis
- Later, the cornea is peripherally thinned out and is called the 'contact lens cornea'
- Due to the collagenase activity, the stromal melt may be more than the epithelial less. As a result, the ulcer may look larger following scraping

Management

1. **Mild, unilateral, without associated collagen vascular disease**
    - Topical corticosteroids with antibiotic cover
2. **Wegener's granuomatosis, polyarteritis nodosa-associated ulcers**
    - Topical cyclosporine A
    - With systemic steroids and immunomodulators
3. **Associated scleritis, bilateral Mooren's ulcer**
    - Systemic immunomodulators
4. **Surgical treatment**
    - Tissue adhesives: Impending perforation
    - Conjunctival resection
    - Tectonic keratoplasty

11. Describe the etiology, clinical features, and management of Mooren's ulcer. (1+2+2 marks)

Mooren's Ulcer

Etiology

- Mooren's ulcer is an idiopathic, severe, autoimmune, peripheral ulcerative keratitis, which spreads circumferentially and centrally to involve the entire cornea
- It is seen more in men, age >60 years, and is bilateral in 30% cases

- The etiology is not known, however, it is thought to be antibodies directed against the stroma of the cornea. Bilateral disease is more aggressive

Clinical Features

Symptoms

- Severe pain
- Lacrimation
- Distorted vision due to astigmatism

Signs

- In the early stages, there is thinning of the superficial third of the peripheral cornea
- The ulcer then progresses circumferentially, with infiltrates ahead of the ulcerative process
- The lagging part of the ulcer shows vascularization at the base
- Complications of this ulcer are perforation following trivial trauma

Management

- It is a difficult ulcer to treat
- Without treatment, the ulcer burns out itself in about a year or so, but the cornea is thinned and vascularized
- Even with topical and systemic steroids, around 20% have very low vision
- Keratoplasty also fails due to recurrence in the donor cornea
  - Topical steroids, ciclosporin, N-acetylcysteine
  - Systemic: Cyclophosphamide and ciclosporin
  - Conjunctival resection: As the immune response brought through the limbal vessels, circumferential conjunctival peritomy may be helpful
  - Keratoplasty along with immunosuppression has some hopeful results

SHORT ANSWERS

1. Enumerate the organisms that can cross the intact epithelial barrier of the cornea. (3 marks)

1. *Corynebacterium diphtheriae*
2. *Neisseria meningitidis*
3. *Neisseria gonorrhoeae*

2. Describe descemetocele. (3 marks)

- The Descemet's membrane is tough
- When the process of ulceration has destroyed the epithelium and the stroma, an extremely thin, focal area of outpouching of the Descemet's membrane is seen
- It is known as descemetocele
- It protects the integrity of the globe, till frank perforation occurs
- It can be seen as infective and noninfective keratitis

3. Enumerate the causes of exposure keratopathy. (3 marks)

1. Facial palsy: Due to Bell's palsy, herpes zoster infection, acoustic neuroma, and parotid tumors.
2. Reduced muscle tone: Parkinsonism, coma
3. Abnormal protrusion of globe: Proptosis of any cause
4. Mechanically restricted eyelids due to scarring, cicatricial pemphigoid, eczema, solar keratosis, and xeroderma pigmentosum

4. Describe the management of exposure keratopathy. (3 marks)

Management of Exposure Keratopathy

- Prophylactic taping of the eyelids during sleep and frequent lubricant instillation is indicated in cases of Facial palsy and proptosis
- A bandage contact lens can be placed to protect the cornea
- Tarsorrhaphy is a method of suturing the upper and lower lids to protect the cornea
- Treatment of the underlying cause for exposure

5. Summarise corneal ulcer scraping. (3 marks)

- Corneal ulcer scraping is a diagnostic method of collecting a sample for microbiological examination from an ulcer
- The patient is seated at the slit-lamp and proparacaine is instilled
- The margins of the ulcer are gently debrided with a Kimura's spatula or a blade after instilling topical anesthesia
- This has a therapeutic effect on the ulcer as unhealthy tissue is removed
- The scrapings from the ulcer are sent for microbiological examination like Gram's stain, KOH mount (for fungi), and are inoculated into blood agar, chocolate agar, and SDA agar
- Care is taken to maintain asepsis as much as possible. The examiner should avoid exposing the plates too much to air or breathing into the plates

6. Enumerate three causes for peripheral corneal ulceration. (3 marks)

1. Rheumatoid arthritis
2. Acne rosacea
3. Wegener's granulomatosis

7. Enumerate three noninfective causes of corneal perforation. (3 marks)

1. Rheumatoid arthritis (most common)
2. Sjogren syndrome
3. Neurotrophic keratopathy

8. The peripheral cornea is predisposed to autoimmunity-induced keratitis. Justify. (3 marks)

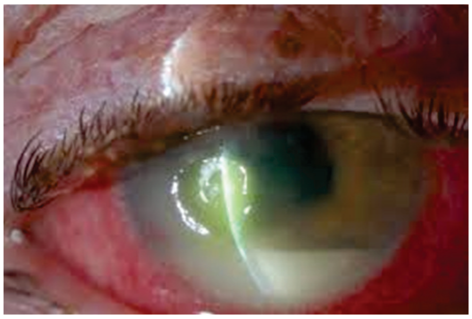
- The peripheral cornea is defined as the part of the cornea outside the central 4–5 mm till the limbus
- This area is significantly different in terms of immunological characteristics which predisposes it to immune-mediated damage
    - Increased concentration of IgM antibodies
    - Increased concentration of C1 complement
    - Presence of blood vessels in the peripheral 0.5 mm and lymphatics that bring in components of the effector and effector arms of the immune response
    - Higher concentration of Langerhans' cells
    - Collection of immune complexes in the peripheral blood vessels leading to complement activation

COMPETENCY

OP4.2 ENUMERATE AND DISCUSS THE DIFFERENTIAL DIAGNOSIS OF INFECTIVE KERATITIS

LONG ESSAYS

1. A 50-year-old manual labourer came to the outpatient department with redness, pain, and mucoid discharge in his right eye of two weeks duration. He gives a history of excess watering the right eye for around two to three years. On examination, the following image is seen.



A. State the most probable diagnosis? (1 mark)

B. Name the most probable organism that is associated with the clinical picture and the relevant ocular history. (1 mark)

C. Explain how you would like to manage this patient. (5 marks)

D. Explain the importance and management of hypopyon in a corneal ulcer. (3 marks)

A. Most Probable Diagnosis

- Bacterial corneal ulcer

B. Most Probable Organism that is Associated with

- With the given history of watering from the affected eye for 2–3 years, the most probable predisposing factor is chronic dacryocystitis
- The most common organism in such cases is *Pneumococcus*

C. Management of this Ulcer

- Ulcer debridement and microbiological examination
  - The margins of the ulcer are gently debrided with a Kimura's spatula or a blade after instilling topical anesthesia
  - This has a therapeutic effect on the ulcer as the scrapings from the ulcer are sent for microbiological examination like Gram's stain, KOH mount (for fungi), and are inoculated into blood agar, chocolate agar, and SDA agar
- Rule out a blocked nasolacrimal duct by doing a lacrimal syringing. If the lacrimal sac is blocked, then an immediate dacryocystectomy should be performed
- Analgesics and anti-inflammatory drugs are given
- Cycloplegics are started right away to relax the ciliary muscle and decrease the pain. It brings down associated uveitis. The drugs that can be used are atropine, homatropine, and tropicamide
- Antibiotics are started after scrapings have been sent for microbiological examination. It is started based on presumptive diagnosis based on the characteristics of the ulcer. Later, based on sensitivity, they can be changed.

Gram-positive cover	Gram-negative cover
• Fluoroquinolones like ciprofloxacin, ofloxacin, gatifloxacin, and moxifloxacin (0.3%) • Cephazolin 75–100 mg/ml • Aminoglycosides like gentamicin (14 mg/ml), Tobramycin (14 mg/ml) • Vancomycin (33 mg/ml)	Aminoglycosides like gentamicin (14 mg/ml), Tobramycin (14 mg/ml)

- Secondary glaucoma is the most common cause of nonhealing ulcers. It may be caused by intense inflammation as evidenced by the formation of hypopyon. Hence, topical beta-blockers or carbonic anhydrase inhibitors are given to bring down IOP. Prostaglandins are contraindicated

D. Hypopyon

Mechanism of Development of Hypopyon

- The ulcerative process leads to violent anterior chamber reaction due to entry of toxins into the anterior chamber
- These exudates and inflammatory cells settle at the bottom in the anterior chamber in the form of hypopyon. It is sterile

Importance

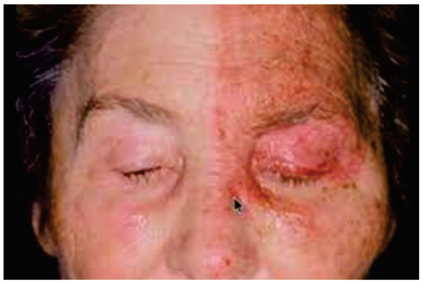
- It is usually sterile in a bacterial corneal ulcer and a reduction in the height of hypopyon should be taken as an improvement of the ulcer
- In fungal keratitis, the hypopyon is more reluctant and need not necessarily be sterile
- The presence of a hypopyon increases the intraocular pressure and may be a cause for delayed healing of the ulcer
- The use of antiglaucoma medications like beta-blockers and carbonic anhydrase inhibitors helps in controlling intraocular pressure.

2. A 44-year-old man presents which severe left temporal pain and ocular pain associated with vesicular lesions on lid margins. Ocular examination reveals conjunctival congestion with poorly staining dendrites.

A. What is the most probable diagnosis? (1 mark)

B. What happens to the corneal sensation in this condition? (1 mark)

C. Describe the natural history, clinical features, and management of this condition. (1+2+5 marks)



A. Most Probable Diagnosis

Herpes zoster ophthalmicus

B. Corneal Sensations are Reduced in Such Cases

C. Herpes Zoster Ophthalmicus

Natural History

- HZO is the infection of the Gasserian ganglion by the varicella zoster virus
- Varicella virus causes chickenpox in childhood and becomes latent in the Gasserian ganglion

- Recurrence is caused due to immunosuppression like HIV, systemic steroids
- The virus gets activated and causes inflammation along the ophthalmic division of the fifth nerve
- During the initial phase, there is severe pain along the ophthalmic division and is followed by the appearance of vesicles over the skin supplied by the same nerve

Clinical Features

- Conjunctiva
  - Resembles viral conjunctivitis, with or without mucopurulent discharge, follicles, and petechial hemorrhages
- Cornea
  - Zoster keratitis is characterized by fine or coarse punctate epithelial keratitis, which later elongates to form microdendrites
  - These are different from the HSV, characterized by shorter branches and the absence of knobs at the ends. Instead, they have tapered ends
  - Nummular keratitis is characterized by a coin-shaped stromal haze of varying sizes. They may coalesce to form disciform keratitis later
  - Complications include metaherpetic keratitis due to loss of sensations and neuroparalytic keratitis due to facial palsy
  - Other manifestations include hypertensive iridocyclitis, episcleritis, and scleritis, acute retinal necrosis, third nerve palsy, optic neuritis, and encephalitis. These occur due to occlusive vasculitis

Management

1. **Systemic therapy**
   i. *Oral antiviral drugs:* They decrease the severity of HZO and the risk of developing postherpetic neuralgia. They should be started immediately after the onset of rash. Acyclovir is the most commonly used drug and is given in a dose of 800 mg 5 times a day for 10 days, or valaciclovir in a dose of 500 mg TDS. It is good to have a baseline LFT before starting acyclovir
   ii. *Analgesics:* NSAIDs and sometimes opioids may be needed depending on the severity of the disease and the patient's pain threshold
   iii. *Systemic steroids:* 40–60 mg per day along with proton pump inhibitors are given to decrease scarring only after starting antiviral therapy. They appear to inhibit the development of postherpetic neuralgia when given in high doses. However, the risk of high doses of steroids in the elderly should always be taken into consideration. Steroids are commonly recommended in cases developing neurological complications such as third nerve palsy and optic neuritis
   iv. *Amitriptyline* should be used to relieve the accompanying depression in the acute phase. It is helpful in postherpetic neuralgia if given in early phases
2. **Local therapy for skin lesions**
   - Antibiotic-corticosteroid skin ointment or lotions
3. **Treatment for eye disease**

Conjunctivitis	Not required if the cornea is not involved
Episcleritis	Resolves spontaneously
Epithelial keratitis	Resolves spontaneously, topical antiviral may be given

Contd.

Stromal keratitis, scleritis, iridocyclitis	Acyclovir 3% ointment five times a day Cycloplegics Topical antibiotics to prevent secondary infections 2 weeks Topical steroids four times a day Topical antiglaucoma medications
Disciform keratitis, nummular keratitis	Topical steroids for prolonged periods, recurrence may occur if stopped early
Neurotrophic keratitis	Bandage contact lenses, lubricants, and eye patching

4. **Postherpetic neuralgia**
    - Topical capsaicin (to deplete substance P) and local anesthetic creams
    - Systemic treatment includes paracetamol, codeine, carbamazepine, and amitriptyline
    - Prophylaxis includes the use of systemic steroids and amitriptyline in the early stages of the disease

SHORT ESSAYS

1. Describe the role of microbiological investigations in infectious keratitis. (5 marks)

Microbiological Investigations in Infectious Keratitis

- The main therapeutic agent for the treatment of infectious keratitis is the right antimicrobial
- Though a clinical diagnosis of the probable organism involved can be guessed from the appearance of the ulcer, a definitive diagnosis is gotten from the microbiological examination
- This is achieved by scraping from the ulcer

Procedure of Scraping

- Corneal ulcer scraping is a diagnostic method of collecting a sample for microbiological examination from an ulcer
- The patient is seated at the slit-lamp, and proparacaine is instilled
- The margins of the ulcer are gently debrided with a Kimura's spatula or a blade after instilling topical anesthesia
- The scrapings from the ulcer are sent for microbiological examination like Gram's stain, KOH mount (for fungi), and are inoculated into blood agar, chocolate agar, and SDA agar
- Special stains like Giemsa, acid-fast stain, acridine orange, calcofluor white, and others are used depending on the clinical scenario
- If the patient is already on antimicrobials, then liquid media can be used to increase the gain of positivity.
- Care is taken to maintain asepsis as much as possible. The examiner should avoid exposing the plates too much to air or breathing into the plates

Uses of Scraping

- It is therapeutic to an extent as it removes the dead and necrotic tissue from the ulcer
- The results obtained help in knowing the type of organism involved and the sensitivity of the organism to a particular drug
- Many organisms may be resistant to empirical treatment. Use of the drugs that the organism is sensitive to, may help in early resolution and healing of the ulcer, and thus better visual outcomes

2. Describe the clinical features of keratomycosis. (5 marks)

Clinical Features of Keratomycosis

Symptoms

- The course is indolent
- Filamentous fungi usually affect immunocompetent individuals whereas yeasts affect immunocompromised individuals. The former is seen more in tropical climate, whereas the latter is seen more in temperate zones
- They are usually mild and as not as dramatic as bacterial corneal ulcers
- Photophobia, blurring vision, and lacrimation are usually seen

Signs

- The ulcer surface is dry-looking, grayish white, with elevated rolled-out margins. In absence of an epithelial defect, there is a localized area of infiltrates with feathery margins and finger-like extensions are present into the surrounding stroma
- A sterile immune ring (yellow line of demarcation) – Wesseley's immune ring may be present where fungal antigen and host antibodies meet
- Multiple, small satellite lesions are characteristic
- A hypopyon is present many a time. It is grossly disproportional to the size of the ulcer and is very dense. Here the hypopyon is not sterile
- It rarely perforates and does not vascularize
- Filamentous fungi usually lead to gray-yellow infiltrate with indistinct margins whereas candida species leads to dense suppuration

3. Outline the manifestations of the herpes simplex virus in the cornea. (5 marks)

Primary Infection

- Can present at 6 months—5 years of age
- It is seen in nonimmune persons

Skin Lesions

Vesicular lesions may occur at lid margins, periorbital area, and the skin of the lids.

Conjunctiva

Acute follicular conjunctivitis with preauricular lymphadenopathy.

Cornea

- 50% of patients have corneal involvement
- Keratitis results due to virus replication and is seen as dendrites (linear branching pattern)
- Stroma is uninvolved

Recurrent Infection

Corneal sensations are decreased due to damage to the Gasserian ganglion.

Punctate Epithelial Keratitis

Seen as fine or coarse epithelial erosions that stain with fluorescein (Fig. 4.2.1)

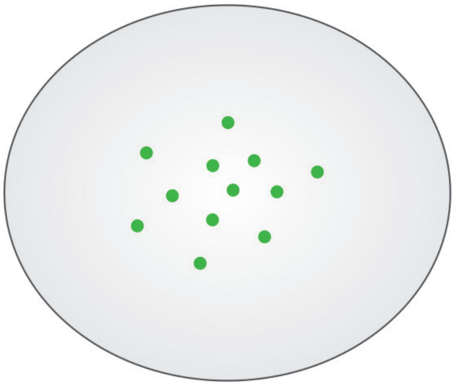


Fig. 4.2.1: Punctae erosions

Dendritic Ulcers

- These are pathognomonic for herpetic infections
- It has a linear branching pattern with terminal bud bed stains with fluorescein (Fig. 4.2.2)
- It is not an ulcer by definition as there are no infiltrates

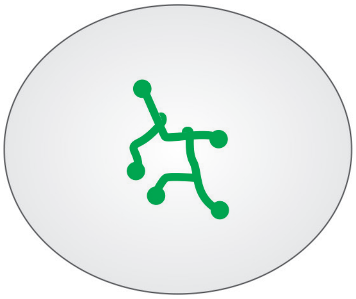


Fig. 4.2.2: Dendrite

Geographic Ulcers

It occurs due confluence of multiple dendrites to form an amoeboid epithelial defect (Fig. 4.2.3)

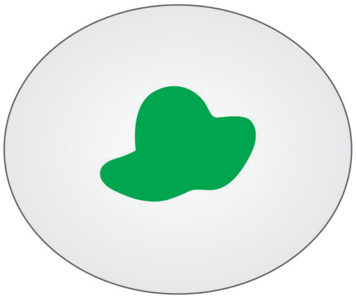


Fig. 4.2.3: Geographic ulcer

Disciform Keratitis

- It is a stromal form of keratitis, due to an immune response mounted against the HSV antigen
- There is inflammation of the endothelium leading to mild stromal edema and Descemet's folds
- Associated features are the presence of keratic precipitates, decreased corneal sensations, and an increase in the IOP

Stromal Necrotic Keratitis

- Interstitial keratitis caused by the active viral invasion and tissue destruction
- Symptoms include pain, photophobia, and redness
- It presents as necrotic, blotchy, cheesy white infiltrates that may lie under the epithelial ulcer
- Mild iritis and keratic precipitates are seen
- It heals by stromal vascularization

Meta Herpetic Keratitis

- It is a type of neurotrophic keratitis, and rather a complication of herpes infection, than the infection itself
- Due to reduced sensations, the epithelial defect is slow to heal and presents as a single oval-shaped defect

4. Describe the clinical features and management of interstitial keratitis. (3+2 marks)

- Interstitial keratitis (IK) is the inflammation of the stroma without involving the epithelium and endothelium
- Though similar in presentation, minor differences are present in three main types of IK

Type of interstitial keratitis	Clinical features	Management
Herpes infection	There is inflammation of the endothelium leading to mild stromal edema and Descemet's folds Associated features are the presence of keratic precipitates, decreased corneal sensations IOP may be raised	Topical antivirals include any of the following (for 2 weeks) Idoxuridine 5% eye drops 5 times a day

Contd.

Type of interstitial keratitis	Clinical features	Management
Syphilis	• Initial stage (first 2 weeks) ▪ Corneal edema due to endothelitis ▪ KPs ▪ Ground glass cornea • Florid stage (2 weeks 2 months) ▪ Deep vascularization gives a salmon patch appearance • Regressive stage ▪ The presence of ghost vessels only ▪ Edema resolves	• Topical steroids • Cycloplegics • Keratoplasty if opacities are dense • Systemic penicillin should be started in the early stages • Systemic steroids in refractory keratitis
Tuberculosis	• Sectoral stromal infiltration with deep vascularization • No associated KPs	• Topical steroids • Cycloplegics • Keratoplasty if opacities are dense • Antitubercular therapy

5. Outline the management of herpetic infections of the anterior segment. (5 marks)

Management of HSV Infections

Epithelial keratitis • Punctate erosions • Dendrites • Geographic ulcers	• Topical antiviral + topical antibiotic + lubricants • Topical antivirals include any of the following (for 2 weeks) ▪ Idoxuridine 5% eye drops 5 times a day ▪ 3% acyclovir ointment 5 times a day ▪ Trifluoridine 1% eye drops 4 times a day
Stromal keratitis	• Topical antivirals as described above + topical prednisolone 1% + cycloplegics +/– antiglaucoma medications for 10 weeks
Iridocyclitis	• Topical antivirals as described above + topical prednisolone 1% + cycloplegics +/– antiglaucoma medications for 10 weeks • Oral acyclovir 400 mg TDS for 10 weeks
Metaherpetic keratitis	• Bandage contact lenses, lubricants • Topical antivirals if associated with infiltrates

Management of Herpes Zoster Ophthalmicus

- Systemic acyclovir 800 mg TID for 2 weeks + anti-inflammatory drugs
- Systemic steroids are indicated for proptosis, optic neuritis, and oculomotor palsy

Conjunctivitis	Not required if the cornea is not involved Topical antibiotic eye drops and ointment	
Episcleritis	Resolves spontaneously	
Epithelial keratitis	Resolves spontaneously, topical antiviral may be given	
Stromal keratitis, scleritis, iridocyclitis	Acyclovir 3% ointment five times a day, Cycloplegics, Topical antibiotics to prevent secondary infections Topical steroids four times a day Topical antiglaucoma medications	2 weeks
Disciform keratitis, nummular keratitis	Topical steroids for a prolonged period, recurrence may occur if stopped early	
Neurotrophic keratitis	Bandage contact lenses, lubricants, and eye patching	

6. Outline the management of fungal keratitis. (5 marks)

Management of Fungal Keratitis

1. Debridement of the ulcer or the overlying epithelium is done and sent for microbiological examination to understand the type of fungi involved. Scrapings are sent for Gram's stain, Giemsa stain, and culture on Sabouraud's dextrose agar. Fungal cultures usually take time to appear and thus broad-spectrum antifungals should be started
2. Cycloplegics
3. Antifungals: Most of the antifungals are fungistatic so, they should be continued for several weeks. They are prescribed as topical, subconjunctival, or systemically. Subconjunctival drugs are reserved for severe keratitis with hypopyon. Systemic drugs are given for severe keratitis and endophthalmitis
4. Yeasts: Econazole, natamycin 5%, amphotericin B 0.15%, clotrimazole
5. Filamentous fungi: Natamycin 5%, econazole, miconazole and amphotericin B 0.15%
6. Systemc antifungals are indicated in deep infections (beyond the Descemet's membrane) and in scleral involvement
7. Broad-spectrum antibiotics should also be given as secondary bacterial infections that can occur
8. Treat the predisposing condition if any
9. Therapeutic keratoplasty may be required in nonresponsive cases

7. Contrast bacterial and fungal keratitis. (5 marks)

Bacterial and Fungal Keratitis

Feature	Bacterial	Fungal
History	Acute onset	Indolent course History of exposure to vegetative matter is usually present
Symptoms	More dramatic, the patient is wincing due to severe pain	Pain is not much
Signs Appearance of ulcer	Wet looking, associated with slough sometimes	Dry looking, epithelium may not be involved in the early stages
Margins	Irregular or regular	Feathery
Other features	Hypopyon may or may not be there Usually sterile	Hypopyon is almost always there Not sterile Presence of satellite bodies Endothelial plaque
Culture	Positive for bacterial	Positive for fungal

8. Describe the etiology and clinical features of ulcus serpens. (2+3=5 marks)

Ulcus Serpens

Etiology

- Ulcus seprens is also called hypopyon corneal ulcer
- The term 'hypopyon corneal ulcer' is reserved for the ulcer caused by *Pneumococcus*. Other ulcers that have hypopyon are called 'corneal ulcer with hypopyon'
- Hypopyon corneal ulcer caused by *Pneumococcus* is called ulcus serpens and the infection is usually transmitted from the coexisting chronic dacryocystitis
- Predisposing factors include debility and alcoholism

Clinical Features

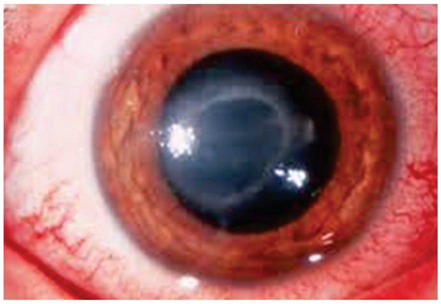
- These patients usually have less pain compared to the ongoing pathology. So, treatment is sought late
- They tend to be more central in location and creep over the cornea in a serpiginous fashion
- The edge of the ulcer that progresses shows surrounding infiltrate whereas, the other edge starts healing
- There is associated hypopyon due to severe iridocyclitis. These ulcers usually increase in size and perforate

9. Describe the various ocular manifestations of the varicella-zoster virus with management. (5 marks)

Varicella-zoster Virus: Ocular Manifestations and Management

Ocular manifestations	Description	Management
Conjunctivitis	Resembles viral conjunctivitis, with or without mucopurulent discharge, follicles, and petechial hemorrhages	Treatment not required if the cornea is not involved
Episcleritis	Can be nodular or diffuse	Resolves spontaneously
Epithelial keratitis	Zoster keratitis is characterized by fine or coarse punctate epithelial keratitis, which later elongates to form microdendrites. These are different from the HSV, characterized by shorter branches and the absence of knobs at the ends. Instead, they have tapered ends	Resolves spontaneously, topical antiviral may be given Systemic antivirals, steroids, and anti-inflammatory drugs to be given as a part of the treatment of HZO
Stromal keratitis, scleritis, iridocyclitis	Iridocyclitis is characterized by stellate KPs, iris atrophy, and ocular hypertension	Acyclovir 3% ointment five times a day, Cycloplegics Topical antibiotics to prevent secondary infections Topical steroids four times a day Topical antiglaucoma medications
Disciform keratitis, nummular keratitis	Nummular keratitis is characterized by a coin-shaped stromal haze of varying sizes. They may coalesce to form disciform keratitis later	Topical steroids for a prolonged period, recurrence may occur if stopped early
Neurotrophic keratitis	Large epithelial defects with or without infiltrates	Bandage contact lenses, lubricants, and eye patching

10. An 18-year old college-going girl presents with severe pain and diminution of vision in her right eye. She gives a history of the use of tap water for cleaning contact lenses. On examination, the following image is seen.



A. What is the most probable diagnosis? (1 mark)

B. How is the diagnosis confirmed? (2 mark)

C. Outline the management of this case? (2 marks)

A. Most Probable Diagnosis

- *Acanthamoeba* keratitis

B. The diagnosis is confirmed by

- Microbiological examination
- Corneal scrapings are mounted with KOH to recognize *Acanthamoeba* cysts
- Calcofluor white stain is specific for acanthamoeba and brightens the cysts bright apple green under a fluorescence microscope
- Culture in a non-nutrient agar which is later seeded with *E. coli* helps isolate *Acanthamoeba*

C. Management

- Cycloplegics
- Debridement
- Antiamoebic drugs like
  - 0.1% propamidine isethionate (Brolene)
  - Neomycin drops
  - Polyhexamethylene biguanide (0.01–0.02% solution)
  - Chlorhexidine
  - These are given in combination
- Ciprofloxacin should be added to cover gram-negative organisms
- The treatment is usually required for a long time (around 3–4 months)
- Oral ketaconazoe 200 mg BD or itraconzole 100 mg OD
- Penetrating keratoplasty is frequently required

11. Enumerate six causes for punctate keratitis. Explain the management of Snow-blindness. (3+2 marks)

Causes of Punctate Keratitis

1. Viral infections like herpes simplex, herpes zoster, and adenovirus
2. Dry eye
3. Associated with staphylococcal blepharoconjunctivitis
4. Chlamydial infection
   - Lagophthalmos and neurotrophic keratitis
5. Vernal keratoconjunctivitis
6. Drugs like beta-blockers, prostaglandins

Management of Snow-blindness/Photo-ophthalmia

- The symptoms of pain and photophobia usually begin 4–6 hours after exposure to UV rays
- Management includes frequent instillation of tear film substitutes, with eye patching with antibiotic ointment for 24 hours
- Oral analgesics if the pain is intolerable

12. Describe the salient results of the herpetic eye disease study. (5 marks)

Herpetic Eye Disease Study (HEDS)

- Was done in two arms: A therapeutic arm and a preventive arm
- It was a series of prospective, double-masked, randomized clinical trials in which the management of herpetic eye disease is defined

- The results of the trials are:
  - HEDS 1
    - This part investigated the treatment of HSV stromal keratitis and iritis
    - Topical trifluridine was used and concluded that topical prednisolone 1% (8 times a day for a week and then tapered over 10 weeks)was found to have much better results in terms of resolution of HSV stromal keratitis
    - There is no apparent benefit of adding systemic antivirals in terms of visual outcome and time of resolution
    - In cases of HSV iridocyclitis, there is a use of adding oral antivirals (400 mg five times a day for 10 weeks) along with topical trifluridine and steroids
  - HEDS 2
    - There is no benefit of adding oral acyclovir to HSV keratitis to reduce the future development of stromal keratitis and iridocyclitis
    - But in patients with a previous history of herpetic eye disease, low dose oral acyclovir (400 mg BID for a year) was found to decrease the rate of recurrence
    - To conclude, a history of HSV epithelial keratitis was not a risk factor for recurrent epithelial keratitis, but previous, especially multiple, episodes of stromal keratitis increased the risk of recurrent stromal keratitis by 10 times

SHORT ANSWERS

1. Describe the management of meta-herpetic keratitis. (3 marks)

- Metaherpetic keratitis is a complication of herpetic eye disease as there is a loss of corneal sensation. Hence, it is a type of neurotrophic keratitis
- Management
  - Stoppage of toxic medications
  - Topical tear film substitutes
  - Bandage contact lens
  - Amniotic membrane grafts
  - Topical steroids are indicated only if there is underlying inflammation

2. Describe the clinical features of herpetic interstitial keratitis. (3 marks)

Clinical Features

Symptoms

Patients' main complaint is visual blurring with or without pain.

Signs

- There is edema with infiltrates in the stroma without any overlying epithelial defect
- It can be localized or diffuse
- There may be a rise in intraocular pressure
- Other features include the presence of keratic precipitates, reduced corneal sensations, the presence of ghost vessels, and deep vascularization

3. Explain the terms

A. Superficial punctate keratopathy (1 mark)

B. Filamentary keratopathy (1 mark)

C. Pannus (1 mark)

A. Superficial Punctate Keratopathy

- Point-sized epithelial defects that stain with fluorescein and rose Bengal caused by a variety of causes
- Superior SPEs are seen in superior limbic keratoconjunctivitis, floppy eyelid, ill-fitting contact lens
- Inferior SPEs are seen in lagophthalmos, blepharitis, drug toxicity
- Interpalpebral SPEs are seen in UV keratopathy, dry eyes, and reduced corneal sensations

B. Filamentary Keratopathy

- Filaments are strands of epithelium with one end stuck to its base and the other mobile with every blink
- They stain with fluorescein
- It is seen in severe dry eye, corneal epithelial instability, superior limbic keratopathy, and neurotrophic keratopathy

C. Pannus

- It is vascularization associated with subepithelial infiltration
- It is seen in cases of chronic inflammation

COMPETENCY

OP4.3 ENUMERATE THE CAUSES OF CORNEAL EDEMA

LONG ESSAYS

1. A child was born with bilateral cloudy corneas with normal corneal diameter.

A. Enumerate the causes for congenital cloudy cornea. (4 marks)

B. Describe the function of endothelium and epithelium in maintaining corneal clarity. (3 marks)

C. Describe the inheritance and clinical presentation of congenital hereditary endothelial dystrophy. (3 marks)

A. Causes of Congenital Cloudy Cornea (Mnemonic STUMPED)

1. **S—Sclerocornea**
    - It is characterized by gradual opacification and vascularization of the cornea
    - May be peripheral (mimics microcornea) or diffuse (involving the whole of the cornea)
2. **T—Tears in the Descemet's membrane**
    - It can occur due to trauma during instrumental delivery leading to hydration of stroma and corneal clouding
3. **U—Ulcers**
    - Ulcerative keratitis may be seen in association with ophthalmia neonatorum, especially with gonococcal infections
4. **M—Metabolic**
    - Mucopolysaccharidoses like Hurler's syndrome, Scheie syndrome, Sanfillipo, and Morquio syndrome are associated with a cloudy cornea
    - Only Hunter's syndrome is unaffected. Others are cystinosis, Fabry's disease, Wilson's disease

5. **P—Peter's anomaly or posterior corneal ulcer**
   - It is a type of anterior segment dysgenesis characterized by partial or total corneal edema and resultant opacification and a central posterior corneal defect with iris strands attached to the cornea and anterior capsular cataract
   - It is also associated with congenital glaucoma
6. **E—Endothelial dystrophy**
   - Congenital hereditary endothelial dystrophy is characterized by bilateral, symmetrical, severe corneal edema at birth due to attenuated endothelial later
   - Other endothelial dystrophies like Fuch's and posterior polymorphous dystrophy present later in life
7. **D—Dermoid**
   - It is seen in association with Goldenhar syndrome

B. Role of Epithelium and Endothelium in Maintaining Corneal Transparency

Role of epithelium	Role of endothelium
• The normal epithelium is transparent due to the homogeneity of the refractive index	• The Na^+–K^+ ATPase pump maintains the relatively dehydrated state of the stroma
• The tight intercellular junctions prevent the entry of the tears from the tear film and act as a barrier function	• Also, the continuity of the endothelium and density of the endothelium helps to cover the Descemet's membrane and prevents the entry of the aqueous • The endothelium has sliding properties that help to keep the continuity

C. Congenital Hereditary Endothelial Dystrophy

Inheritance	Clinical presentation
CHED 1: Autosomal dominant	• Presents in the first or second decade • In asymptomatic cases, the moon crater-like appearance of the endothelium • Later on, visual acuity is typically less on awakening • Corneal clouding ranging from a diffuse haze to ground glass milky appearance • Corneal thickness is twice the normal • The IOP is usually normal
CHED 2: Autosomal recessive	• Presents at birth • Similar to CHED 1 • Severity is more • May be associated with nystagmus

2. Normally, the cornea is a transparent structure.

A. Define corneal edema. (1 mark)
B. Describe the causes of corneal edema. (3 marks)
C. Describe symptoms and signs of corneal edema. (3 marks)
D. Enumerate treatment modalities for corneal edema. (3 marks)

A. Corneal Edema

- An increased presence of fluid within the cornea
- It can be in the epithelium, stroma, or both

B. Causes of Corneal Edema

- Raised intraocular pressure due to glaucoma
- Endothelial dysfunction
  - Trauma and loss of endothelium due to either intraocular surgery or external trauma (aphakic and pseudophakic bullous keratopathy)
  - Various types of inflammation like uveitis, keratitis, and endophthalmitis, Fuch's heterochromic iridocyclitis
  - Dystrophies such as Fuch's endothelial dystrophy, posterior polymorphous dystrophy, congenital hereditary endothelial dystrophy, stromal dystrophies like lattice dystrophy, granular dystrophy
- Epithelial dysfunction due to chemical injuries, mechanical injuries, and burns

C. Symptoms and Signs of Corneal Edema

Symptoms

- Colored halos (in early stages)
- Diminution of vision
- Pain (when associated with increased IOP)

Signs

- The stroma is hazy, with a loss of transparency and Descemet's folds
- A further rise in intraocular pressure causes microcystic epithelial edema
- Long-standing edema causes vesicles to appear in the epithelium
- These coalesce to form bullae and lead to bullous keratopathy

D. Management

- Management of corneal edema involves identification of the underlying cause. A thorough evaluation is necessary
- Confocal microscopy is performed to study the endothelium (as specular microscopy cannot give clarity on the health of endothelium in a hazy cornea)
- Other investigations include measurement of IOP (using applanation or tonopen) and corneal thickness-pachymetry (ultrasound or OCT)
- Lower IOP if that is the cause. It can be done by topical antiglaucoma medications and systemic drugs
- Hypertonic saline helps mild edema by removal of fluid by osmosis. They are available as topical eye drops and ointments
- Replace endothelium if the endothelial count is caused by Descemet stripping automated endothelial keratoplasty (DSAEK) or Descemet's membrane endothelial keratoplasty (DMEK)
- Penetrating keratoplasty for a scarred cornea

SHORT ESSAYS

1. Explain the factors that help in maintaining corneal transparency. (5 marks)

Factors Maintaining Corneal Transparency

1. **Role of precorneal tear film**
   - A healthy precorneal tear film is also an important component for maintaining corneal transparency

2. **Role of epithelium**
    - The normal epithelium is transparent due to the homogeneity of the refractive index.
    - The tight intercellular junctions prevent the entry of the tears from the tear film and act as a barrier function: Evaporation of water from the ocular surface. The cornea is avascular. Normally, blood vessels enter not more than 1 mm into the peripheral cornea. The rest of the cornea is avascular
3. **Role of stroma**
    - Avascularity of the stroma
    - Uniform alignment of lamella (destructive interference of scattered light) and fibers are smaller than the wavelength of light because of which they do not interfere with light
    - Nerves do not have a myelin sheath
4. **Role of endothelium**
    - The Na^+–K^+ ATPase pump in the endothelium maintains the relatively dehydrated state of the stroma
    - Also, the continuity of the endothelium and density of the endothelium helps to cover the Descemet's membrane and prevents the entry of the aqueous
    - The endothelium has sliding properties that help to keep the continuity
    - The endothelium also has a carbonic anhydrase pump that helps to restrict the entry of fluid from the aqueous into the stroma

2. What is the normal endothelial count? Describe the normal endothelium as seen on specular microscopy. Explain specular microscopy findings of Fuch's endothelial dystrophy. (1+2+2 marks)

Normal Endothelial Count

4000 cells/mm^2 and it decreases with age.

Normal Endothelium on Specular Microscopy

- The endothelium is seen as hexagonal and dark cells
- It shows the number of cells/mm^2

Age	Avereage cell density (cells/mm^3)
10 – 19	2,900–3,500
20 – 29	2,600–3,400
30 – 39	2,400–3,200
40 –49	2,300–3,100
50 –59	2,100–2,900
60 –69	2,000 –2,800
70 –79	1,800–2,600
80 –89	1,500–2,300

- It also shows various values like
    - Cell area
    - Cell density
    - Polymegathism
    - Pleomorphism (hexagonality >70–80%)
    - Coefficient of variance (<0.25)
    - Central corneal thickness

Endothelium in Fuch's Endothelial Dystrophy

- Thickening of the endothelium and presence of excrescences called guttae
- Guttae appear as focal thickening and projections of size around 5 times the size of a regular cell
- A decreased endothelial count, loss of hexagonality of the endothelium, and development of guttae
- In severe disease, there is a confluence of guttae and thickening of the Descemet's membrane

3. Describe the clinical features and management of Fuch's endothelial dystrophy. (5 marks)

Fuch's Endothelial Dystrophy

Clinical Features

- Fuch's endothelial dystrophy is an autosomal dominant condition and presents at the age of 50–60 years, a bilateral decrease in the endothelial count
- On slit-lamp examination
  - Corneal guttata (excrescences) of descemet membrane
  - Stromal edema due to endothelial decompensation
  - Epithelial edema and bullous keratopathy
  - Corneal scarring

Management

- Bandage contact lens
- Hypertonic saline drops
- Using warm air blowers to decrease edema
- Endothelial keratoplasty (Descemet's membrane endothelial keratoplasty and Descemet's stripping automated endothelial keratoplasty)

SHORT ANSWERS

1. Describe the clinical features and treatment of pseudophakic bullous keratopathy. (3 marks)

Pseudophakic Bullous Keratopathy

Clinical Features

- It is seen following intraocular surgery and most commonly follows cataract surgery
- It usually manifests around 4–6 weeks following surgery
- Patients present with diminished vision, tears, and pain
- On examination, there is stromal edema, along with epithelial and subepithelial bullae

Treatment

- Topical hypertonic saline
- Topical anti-inflammatory drugs
- Topical antiglaucoma medications
- Systemic L-cysteine (experimental)
- Penetrating keratoplasty
- Corneal collagen crosslinking to temporarily decrease edema
- Phototherapeutic keratectomy (to relieve pain)

2. Explain the terms regarding corneal edema

A. Bulla	(1 mark)
B. Vesicles	(1 mark)
C. Striate keratopathy	(1 mark)

A. Bulla

- Accumulation of fluid between basal epithelial cells of cornea cause blister-like formation
- They are formed by the coalescence of vesicles

B. Vesicles

Accumulation of minute volumes of fluid in the tight intercellular spaces in the epithelium.

C. Striate Keratopathy

It is a common postoperative complication characterized by folds along the Descemet's membrane and associated with stromal edema.

COMPETENCY

OP4.4: ENUMERATE THE CAUSES AND DISCUSS THE MANAGEMENT OF DRY EYE

LONG ESSAY

1. A 46-year-old woman with rheumatoid arthritis complains of a pricking sensation in both eyes on examination ocular surface appears dry.

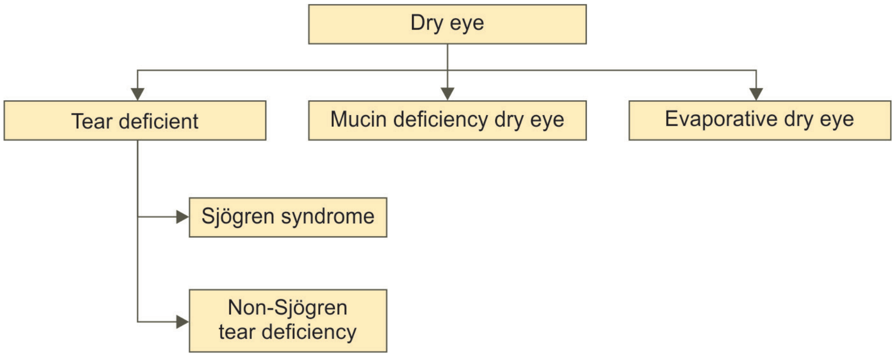
A. Define dry eye.	(1 mark)
B. Classify dry eye.	(3 marks)
C. Explain the diagnostic tests used in dry eye.	(3 marks)
D. Describe the management of dry eye.	(3 marks)

A. Definition of Dry Eye

A disorder of the tear film characterized by decreased tear production or excessive tear evaporation leading to damage to the interpalpebral ocular surface and is associated with subjective symptoms of ocular discomfort.

B. Classification of Dry Eye

Dry eye is classified based on the layer which is pathological in the tear film.



Tear deficient	***Sjögren syndrome*** • It is an autoimmune condition, due to lymphocytic damage to the lacrimal gland acini, due to which there is dry eye • Also, there is associated dry mouth (xerostomia) • It is called primary when it is not associated with any connective tissue disorder • Secondary Sjögren syndrome is usually associated with a connective tissue disease like RA, SLE, scleroderma, polyarteritis nodosa, and Wegener's granulomatosis ***Non-Sjögren tear deficiency*** Can arise due to disease of the lacrimal gland or obstruction of the ducts or due to loss of reflex arc for tear production
Mucin deficient	• Occurs due to destruction of goblet cells due to hypovitaminosis A, cicatrizing ocular surface disorders like Stevens–Johnson syndrome
Evaporative dry eeye	• Meibomian gland disease: the outer part of the tear film is deficient and hence tears get evaporated easily • Bell's palsy and other causes of facial palsy • Preservatives used for drug formulations have detergent-like action on the lipid layer and thus lead to dry eye. The most common preservative associated is BAK- benzalkonium chloride • Low blink rate, as seen individuals constantly working on the computer

C. Diagnostic Tests Used for Dry Eye

1. **Tear film height**
   - It is tested by instilling a drop of fluorescein in the conjunctival sac
   - Tear film height should be around 1 mm at the inferior lid margin
   - In dry eyes, it is lesser
2. **Tear film break up time**
   - After instilling fluorescein, the patient is asked to blink and then keep eye open
   - The eye is examined under blue light
   - The time taken for the first dry patch to appear on the cornea is noted
   - Normally, it is around 15–30 seconds
   - Lesser than 10 seconds is suggestive of dry eye
   - It detects all types of dry eye
3. **Schirmer I test**
   - Whitman-41 filter paper of dimensions around 35 mm × 5 mm is placed at the junction of lateral third and medial two third of the lower lid margin
   - The patient is asked not to blink and look straight ahead (upgaze increases palpebral aperture hence increases drying of tear film) for 5 minutes
   - Normal values are more than 15 mm, <10 mm is moderate dry eye and <5 mm is severe dry eye
   - It measures the aqueous function of the tear film
4. **Schirmer II and III test**
   - In Schirmer II, topical anesthesia is instilled and then test is repeated/
   - Schirmer III test is performed by nasal mucosal stimulation.
5. **Others**
   - Corneal sensations
   - Rose Bengal staining
   - Antibodies for detecting Sjögren's syndrome: Anti-RO, Anti-La, antisalivary protein 1

  - Tear osmolarity
  - Conjunctival impression cytology

D. Management of Dry Eye

Identify the cause and treat if possible.

Aqueous Deficiency

- Tear film substitutes like carboxymethyl cellulose, polyvinyl alcohol, hypromellose, etc
- Topical cyclosporine can be tried in inflammatory causes for dry eye
- Mucolytics like 5% acetylcysteine are given to decrease mucous strands
- Punctal plugs
  - Silicone plugs are inserted into the lower puncta and the progress of dry eye is re-evaluated
  - If it is found beneficial, then electrocauterization can be done as a permanent measure
  - Topical retinoids are being tried

Meibomian Gland Disease

- Lid hygiene, lid scrubs, warm compresses, and topical azithromycin
- Oral doxycycline is useful in acne rosaceae and seborrhoeic dermatitis
- Tarsorrhaphy for facial palsy
- Zidovudine is helpful in primary Sjögren syndrome
- Submandibular gland transplantation

Mucin Deficiency (if Associated with Vitamin A Deficiency)

- Vitamin A supplementation
- Tear film substitutes

SHORT ESSAYS

1. Explain how vitamin A deficiency causes dry eye and the management of the same. (5 marks)

Or

A 1-year-old child living in a slum presents with night blindness and corneal xerosis following an episode of measles. Explain the probable cause for this clinical scenario. How will you manage this patient? (1+4 marks)

- The most probable cause is vitamin A deficiency that is precipitated by measles
- Vitamin A deficiency causes night blindness and corneal xerosis

Role of Vitamin A in Dry Eye

- Vitamin A is required for adequate health of the goblet cells of the conjunctiva and the cornea
- The goblet cells are involved in producing mucin which forms the innermost layer of the tear film
- This layer is responsible for wetting the conjunctival and corneal surfaces
- The deficiency of this layer causes the inability of the tear film to maintain continuity
- Vitamin A deficiency is usually seen malnourished children and is precipitated by illnesses like measles in childhood
- These children present with bilateral corneal xerosis and ulceration

- The result of vitamin A deficiency is a dry ocular surface seen as conjunctival xerosis (stage X1A), Bitot spots (stage X1B), and corneal xerosis (stage X2)
- Conjunctival xerosis is seen as wrinkling of the conjunctiva in the interpalpebral area
- Bitot spots result due to squamous metaplasia of the conjunctival epithelium (the mucosa becomes more like skin)
- This dryness leads to the proliferation of *Corynebacterium xerosis* which produces a foam-like secretion that covers the brown dry conjunctiva that gives the conjunctiva a silvery appearance

Management of Dry Eye in Vitamin A Deficiency

- Nutritional counseling
- Topical tear film substitutes in the form of HPMC, hypromellose
- Systemic vitamin A supplementation

Oral vitamin A	• >1 year: 2 lakh IU on days 0,1, and 28 • <1 year: 1 lakh IU on days 0,1, and 28
Intramuscular vitamin A (indicated in severe vomiting, diarrhea)	• >1 year: 1 lakh IU on days 0,1, and 28 • <1 year: 50,000 IU on day 0,1, and 28
Women of reproductive age	• For conjunctival xerosis and Bitot spots: oral vitamin A 1 lakh IU daily for 2 weeks • For corneal xerosis: oral vitamin A 2 lakh IU daily for 2 weeks

2. A 19-year-old techie complains of a pricking sensation in both eyes at the end of the day associated with redness.

A. State the type of dry eye seen in this profession? (1 mark)

B. Explain the clinical features and management of this condition. (2 marks)

C. Describe two investigations used for the assessment of dry eyes. (2 marks)

A. Type of Dry Eye Seen

Evaporative dry eye

B. Clinical Features and Management

Clinical Features

Symptoms

- Foreign body sensation
- Ocular irritation
- Lacrimation
- Redness

Signs

- Interpalpebral congestion
- Decreased tear film height
- Increased tear film break-up time
- Normal corneal sensations

Management

Lifestyle modifications

- Decrease in screen time
- Increase in blink rate
- Decreasing the use of directly blowing air to the face (to prevent evaporation of tears)

Medications

- Tear film substitutes
- Punctal plugs in severe dryness

C. Investigations Used for Dry Eye

1. **Tear film height**
    - It is tested by instilling a drop of fluorescein in the conjunctival sac
    - Tear film height should be around 1 mm at the inferior lid margin
    - In dry eyes, it is lesser
2. **Tear film break-up time**
    - After instilling fluorescein, the patient is asked to blink and then keep eye open
    - The eye is examined under blue light
    - The time taken for the first dry patch to appear on the cornea is noted
    - Normally, it is around 15–30 seconds
    - Lesser than 10 seconds is suggestive of dry eye
    - It detects all types of dry eyes

3. Describe the etiology for tear deficient dry eye. (5 marks)

Sjögren-associated Tear Deficiency

- It is an autoimmune condition, due to lymphocytic damage to the lacrimal gland acini, due to which there is dry eye
- Also, there is an associated dry mouth
- It is called primary, when it is not associated with any connective tissue disorder
- Secondary Sjögren syndrome is usually associated with a connective tissue disease like RA, SLE, scleroderma, polyarteritis nodosa, and Wegener's granulomatosis

Non-Sjögren-associated Tear Film Deficiency

- Can arise due to disease of the lacrimal gland or obstruction of the ducts or due to loss of reflex arc for tear production like
    - Primary alacrimia, hypoplastic or aplastic lacrimal gland
    - Secondary to infiltration of lacrimal gland sarcoidosis, HIV, lymphoma, ablation of lacrimal gland
    - Mechanical trauma to efferent secretomotor fibers
    - Graft versus host response following hematopoietic stem cell transfer
    - Systemic medications like anticholinergics, topical alpha agonists, beta-blockers
    - Obstruction to ducts can occur due to scarring from Stevens–Johnson syndrome, trachoma, pemphigoid, and chemical injuries
- Neurotrophic keratopathy, diabetic neuropathy due to decrease in corneal sensations
- Use of contact lens—decrease in reflex secretions
- Following refractive surgeries due to damage to corneal nerves

SHORT ANSWERS

1. Describe the principles and indications of silicone punctal plugs. (3 marks)

Silicone Punctal Plugs

Principles

- The use of punctal plugs for dry eyes is a simple, safe, and reversible procedure. They act by reducing the tear film drainage from the nasolacrimal system

- Initially, temporary plugs are placed in the punctum and the improvement is noted. These are made of absorbable material (collagen). If there is improvement in the dry eye condition, then permanent plugs (made of silicone) are placed

Indications

1. A moderate and severe dry eye that does not respond to topical drugs
2. Schirmer test <5 mm

2. Describe the procedure of punctal occlusion. (3 marks)

Procedure

It can be done in two methods: Temporary and permanent.

Temporary Occlusion

- It is an outpatient procedure
- Temporary occlusion is achieved by inserting collagen plugs in the lower punctum as they get dissolved in a few weeks
- The patient is reviewed after 2–3 weeks to look for improvement of dry eye symptoms
- Long-term temporary occlusion can be achieved by inserting silicone plugs into the canaliculi

Permanent Occlusion

- It is achieved either by thermal or electric cautery and occluding the puncta
- This procedure is contraindicated in the young and in reversible causes of dry eye

3. Describe the principle of using contact lenses as a therapeutic option in dry eyes.

Or

Justify the use of contact lens as a therapeutic option for dry eye (3 marks)

- Contact lenses are known to cause dry eye by decreasing reflex secretion
- However, high water content lenses are useful in dry eyes by causing the fluid to be trapped behind the contact lenses that act as a tear reservoir
- These lenses are available as daily disposable contact lenses and are made up of silicone hydrogel
- Also, they protect the cornea from secondary changes like persistent epithelial defects and help healing faster
- Scleral contact lenses also can be used

4. Enumerate three nonconventional therapy for dry eyes. (3 marks)

1. Salivary gland transplantation
2. Botulinum injection into the orbicularis muscle
3. Autologous blood eyedrops

5. Summarise the clinical features of Meibomian gland dysfunction. (3 marks)

Clinical Features of Meibomian Gland Dysfunction

Symptoms

- Ocular irritation
- Foreign body sensation
- Photophobia

Signs

- Increased tear film debris
- Thickened meibomian secretion
- Capping of meibomian orifices
- Decreased tear film height

COMPETENCY

OP4.5 ENUMERATE THE CAUSES OF CORNEAL BLINDNESS

SHORT ESSAYS

1. Enumerate four causes for corneal blindness. Explain the role of Vitamin A deficiency in corneal opacity. (2+3 marks)

Causes of Corneal Blindness

1. Ulcerative keratitis and infectious keratitis: Bacterial, fungal and acanthamoeba keratitis in decreasing order of incidence
2. Pseudophakic bullous keratopathy: Can occur during phacoemulsification or SICS
3. Dystrophies: Fuch's endothelial dystrophy, CHED, posterior polymorphous dystrophy, stromal dystrophies like granular dystrophy, lattice dystrophy, and macular dystrophy
4. Keratectasias like keratoconus, keratoglobus, and post-LASIK ectasia vitamin A deficiency and corneal opacity

Role of Vitamin A Deficiency

- Vitamin A has two prominent functions in the eye: The production of retinol that is involved in the visual pathway and the second is maintaining the health of the goblet cells of the conjunctiva and the cornea
- Vitamin A deficiency is associated with conjunctival xerosis, Bitot spots, corneal xerosis, corneal ulcer formation, and finally keratomalacia
- Due to vitamin A deficiency, there is a decrease in mucin production by the goblet cells. As a result, the tear film formed is inadequate. Also, the cell repair process of the epithelium is affected
- Vitamin A deficiency-associated corneal ulcers are quick to respond to systemic vitamin A

2. Classify corneal dystrophies and explain clinical features of one of each type of epithelial, stromal, and endothelial dystrophy. (2+3 marks)

Classification of Corneal Dystrophies

Epithelial

1. Map-dot-fingerprint dystrophy
2. Reis–Buckler's dystrophy
3. Meesman's dystrophy
4. Recurrent corneal erosion syndrome
5. Stocker–Holt dystrophy

Stromal

1. Granular dystrophy
2. Lattice dystrophy
3. Macular dystrophy
4. Crystalline dystrophy

Endothelial

1. Fuch's endothelial dystrophy
2. Congenital hereditary endothelial dystrophy
3. Posterior polymorphous dystrophy

Clinical Features

1. **Map-dot-fingerprint dystrophy**
    - It is common dystrophy, seen usually in adults with sporadic distribution occurring due to loss of hemidesmosome between the basement membrane and the epithelium
    - These individuals present with dot-like or fingerprint pattern-like opacities in the anterior part of the cornea
    - As a result, the cornea is very susceptible to erosions
    - Symptoms are mild pain and ocular irritation, and on examination, one can find multiple dot-like or map or fingerprint-like opacities, and due to this varied appearance, it is often misdiagnosed
2. **Granular dystrophy**
    - Patients present with photophobia and lacrimation due to frequent erosions. Visual acuity decreases as the opacification progresses
    - On examination, there are small, white granular opacities in the stroma of the central cornea, surrounding corneal haze, limbus is spared
    - Initially, these granular deposits are seen in the anterior stroma but later they spread to the deeper parts of the stroma and the Descemet's membrane
3. **Fuch's endothelial dystrophy**
    - Presents at the age of 50–60 years, bilateral decrease in endothelial count
    - Patients present with a blurring of vision in the early stages, especially in the morning. There may be associated colored halos
    - Corneal guttata (excrescences) of Descemet's membrane
    - Stromal edema due to endothelial decompensation
    - Epithelial edema and bullous keratopathy
    - Corneal scarring

3. Describe the management of corneal opacity. (5 marks)

Management of Corneal Opacity

- Opacification of the cornea is an irreversible process and thus requires replacement from a clear cornea
- This process is called optical keratoplasty, wherein the keratoplasty is performed for an optical condition

1. **Keratoplasty**
    - The types of keratoplasty are:
        - Penetrating keratoplasty: Most commonly performed, usually for opacities involving half to full thickness
        - Anterior lamellar keratoplasty: For pathologies involving the anterior half of the cornea
        - Descemet's membrane endothelial keratoplasty: For diseases involving the endothelium
2. **Phototherapeutic keratectomy**
    - This technique uses excimer laser to treat superficial corneal opacities

  - In this procedure, the epithelium is gently debrided, and then HPMC (hydroxypropyl methylcellulose) is applied
  - The irregularities protrude out through the smoothness created by the HPMC. This is ablated using excimer LASER

3. **Optical iridectomy**
   - It is a traditional procedure and hardly followed nowadays
   - An iridectomy is created in the area outside the corneal opacity to allow passage of light and some useful vision
4. **Cosmetic procedures**
   - Corneal tattooing and colored contact lenses are used when other options are not possible
   - Corneal tattooing is performed after debriding the epithelium and blotting the stroma with gold chloride solution or platinum chloride solution. These methods are meant to hide the unsightly scar with no optical advantages

SHORT ANSWERS

1. Define corneal opacity. Classify corneal opacity based on its appearance. (3 marks)

Corneal Opacity

A permanent loss of transparency of a part or whole of the cornea due to scarring

Classification of Corneal Opacity

Nebula	Mild opacification, iris details are visible. involves superficial layers of stroma and Bowman's membrane
Macula	Moderate opacification, iris details are not visible, but iris is visible Involves half of the stroma
Leucoma	Dense opacification, underlying structures not visible Scarring involves more than half of the stroma

2. Explain how onchocerciasis causes corneal opacification.

Or

A Nigerian worker presents with bilateral corneal opacity. He says he was diagnosed with "river blindness". Explain how, the etiological agent causes corneal opacity. (3 marks)

- Onchocerciasis is a helminthic infestation caused by the worm *Onchocerca volvulus*. It is common in the regions along the river Nile, and so, is also called river blindness
- The microparasites enter the bloodstream through the bite of the Simulium black fly. The microfilariae enter various tissues and bring about their manifestations
- These microfilariae enter the aqueous through the bloodstream and cause uveitis. Also, they enter the corneal stroma
- In the cornea, there is an immune response mounted against these dead microfilariae and thus produces snowflake opacities
- Slowly the infiltrates involve the whole of the cornea producing sclerosing keratitis. This leads to a vascularized opaque cornea

3. Explain the management of post-adenovirus corneal opacity. (3 marks)

- Adenoviral conjunctivitis, especially epidemic keratoconjunctivitis (EKC) is associated with punctate infiltrates in the cornea
- These tend to persist for long periods and cause dispersion of light and blurred vision

Management

- They are treated with topical steroids (like loteprednol, fluoromethanol) initially for the first week after the active infection is controlled
- This is followed by the use of topical tacrolimus and cyclosporine for around 3 months till the infiltrates disappear

4. Explain the terms

A. Corneal facet (1 mark)
B. Dellen (1 mark)
C. Adherent leukoma (1 mark)

A. Corneal Facet

Depression at the site of a scar on the cornea is called a corneal facet.

B. Dellen

- A shallow saucer-like excavation in the periphery of the cornea
- It occurs due to localized tear film instability

C. Adherent Leukoma

Incarcerated iris into a healed opacity of the cornea is called an adherent leukoma.

5. Describe cornea verticillata and give two causes. (1+2 marks)

Cornea Verticillate

- Also known as vortex keratopathy due to the appearance of whorl-like opacities in the corneal epithelium
- They usually do not give any visual symptoms and the whorl-like pattern is in the direction of migration of corneal epithelial cells
- Rarely it can cause glare

Causes

1. Fabry's disease
2. Systemic amiodarone

6. Vitamin A is essential to the clarity of the cornea. Is this statement true? If so, justify your statement. (1+2 marks)

Yes, this statement is true

Justification

- Vitamin A has two prominent functions in the eye: The production of retinol that is involved in the visual pathway and the second is maintaining the health of the goblet cells of the conjunctiva and the cornea

- Vitamin A deficiency is associated with conjunctival xerosis, Bitot spots, corneal xerosis, corneal ulcer formation, and finally keratomalacia
- Due to vitamin A deficiency, there is a decrease in mucin production by the goblet cells. As a result, the tear film formed is inadequate. Also, the cell repair process of the epithelium is affected
- The integrity of the epithelial cells is also affected

COMPETENCY

OP4.6 ENUMERATE THE INDICATIONS AND THE TYPES OF KERATOPLASTY

SHORT ESSAYS

1. Define keratoplasty and explain the types of keratoplasty? (1+4 marks)

Keratoplasty/Corneal Grafting

Means replacing the host cornea with a donor cornea.

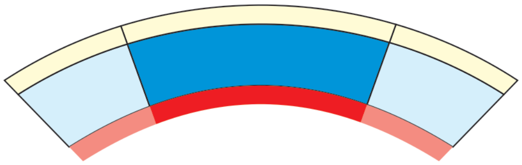


Fig. 4.6.1: Penetrating keratoplasty

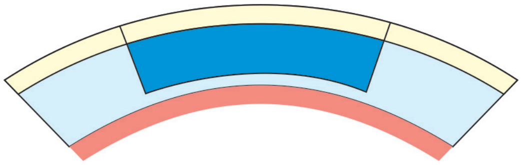


Fig. 4.6.2: Anterior lamellar keratoplasty

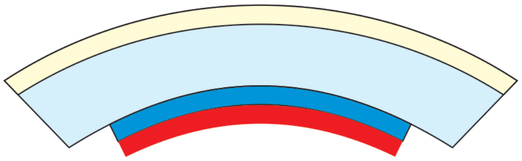


Fig. 4.6.3: Descemet's stripping automated endothelial keratoplasty

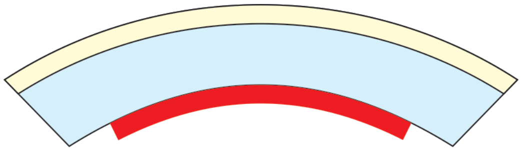


Fig. 4.6.4: Descemet's membrane endothelial keratoplasty

Types of Keratoplasty

- Depending on the depth
  1. **Penetrating keratoplasty (Fig. 4.6.1)**
     - Full-thickness corneal graft
  2. **Lamellar keratoplasty (Fig. 4.6.2)**
     - Anterior lamellar
       - ♦ It is performed when the endothelium and the Descemet's membrane are intact
       - ♦ Depending on the depth of dissection, it may be superficial or deep
     - Posterior lamellar
       - ♦ Deep lamellar endothelial keratoplasty
         a. Descemet's stripping automated endothelial keratoplasty (Fig. 4.6.3)
         b. Descemet's membrane endothelial keratoplasty (Fig. 4.6.4)
  3. **Patch graft**
     - It is used for localized corneal opacities
     - It can be full thickness or partial thickness
- Based on Indication
  1. **Optical keratoplasty**
     - To improve visual acuity in scarred corneas—it may be due to infective ulceration, pseudophakic bullous keratoplasty, dystrophies, trachomatous scarring, or keratomalacia
  2. **Therapeutic keratoplasty**
     - To remove the pathology as in nonresponsive infective keratitis. A fungal corneal ulcer is the most common indication

3. **Tectonic keratoplasty**
    - To restore the integrity of the globe, as in following perforation of corneal ulcer or formation of a corneal fistula
4. **Cosmetic keratoplasty**
    - To improve the appearance of the eye

2. Demonstrate the understanding of evaluation of an eye before keratoplasty. Describe the procedures of management of endothelial dysfunction leading to corneal opacity. (3+2 marks)

Evaluation of an eye before Keratoplasty

- A patient planned for keratoplasty should undergo a complete ophthalmological evaluation to define the outcomes of the surgery
- Evaluation includes
    - Best-corrected visual acuity
        - The patient should have at least PL+, PR accurate vision before penetrating keratoplasty
        - If it is better than PL+, it must be noted clearly
    - A careful slit-lamp examination is done to rule out
        - Corneal vascularization (poor outcome)
        - Ocular inflammation (must be treated before surgery)
        - Blepharitis (to minimize ocular surface infection)
        - Lid abnormalities like entropion, ectropion, lagophthalmos, and trichiasis
        - Dry eyes
        - Other abnormalities like rosacea also must be ruled out
    - Pupil examination of the other eye helps rule out reverse RAPD
    - Intraocular pressure measurement is a challenge in scarred corneas with Goldmann applanation tonometry. Other methods like Tonopen can be used. IOP must be well controlled before surgery
    - Fundus examination may not be possible due to an opaque cornea. Hence a B-scan is mandatory to rule out retinal detachment
    - An ultrasound biomicroscopy can help detect other anterior segment abnormalities

Procedures for Corneal Opacity Due to Endothelial Dysfunction

1. **Penetrating keratoplasty**
    - This is indicated when the opacity of full thickness of the cornea and the whole cornea is replaced by a donor cornea with a healthy endothelium
2. **Descemet's membrane keratoplasty and Descemet's stripping automated endothelial keratoplasty**
    - In this procedure, only the endothelium with Descemet's membrane is transplanted and an air bubble is used to maintain it against the cornea

3. Describe the procedure of penetrating keratoplasty. (5 marks)

Procedure of Penetrating Keratoplasty (Fig. 4.6.5)

- The eye is prepared for surgery by decreasing the IOP by giving intravenous mannitol
- Topical miotics are instilled to constrict the pupil and protect the lens during the surgery
- The size of the donor graft is usually 0.25–0.5 mm larger than the recipient tissue

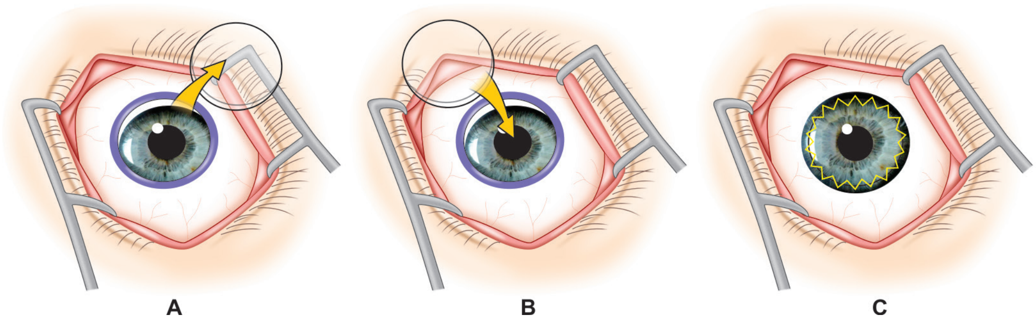


Fig. 4.6.5: Steps of penetrating keratoplasty

- In cases of aphakic eyes, the donor button is 0.5 mm more than the recipient, and in keratoconus, the same size as the recipient
- An inked radial keratotomy marker is used to make marks on the peripheral cornea on the recipient bed
- The donor cornea is cut from the endothelium side
- The recipient cornea is cut using a trephine. Femtosecond LASER can also be used. The trephination is done with partial-thickness followed by a controlled entry into the anterior chamber
- The viscoelastic substance is injected into the anterior chamber
- The recipient corneal button is excised with a corneoscleral knife and the edges are made perpendicular
- Peripheral iridectomy is performed
- The donor corneal button is placed on the recipient bed and 4 cardinal sutures are placed using 10–0 nylon.
- The depth of sutures should be 90% of the depth of the corneal tissue
- The remaining sutures are placed in an interrupted or continuous fashion
- After suturing is complete, all the sutures are buried and the anterior chamber is formed using saline

4. Explain five important postoperative complications of keratoplasty. (5 marks)

Postoperative Complications of Keratoplasty

1. **Flat anterior chamber**
    - The most common cause for a flat anterior chamber post-keratoplasty is wound leak
    - It is associated with hypotony and can be easily identified using Siedel's test and resuturing is done in the operating room.
    - Other causes are:

Cause	Features	Management
Pupillary block	High IOP, a flat peripheral anterior chamber with a deep center	Treated with steroids and cycloplegics YAG iridotomy
Malignant glaucoma	High IOP, diffusely flat anterior chamber	Cycloplegics, Nd YAG hyaloidotomy
Ciliochoroidal detachment	Very low IOP, diffusely flat IOP Kissing choroidal on B-scan	Topical steroids and cycloplegics
Suprachoroidal hemorrhage	High IOP, diffusely flat anterior chamber B-scan is diagnostic: It shows a dome-shaped hemorrhagic choroidal detachment	Intravenous mannitol Sclerotomies to drain the blood

2. **Persistent epithelial defect**
    - The probable causes for a persistent epithelial defect are the toxicity of topical medications, preexisting ocular surface disorders, and suture associated abrasions
    - They are treated with lubricants and decreasing other topical medications
3. **Postoperative endophthalmitis**
    - May result due to contaminated donor tissue, exogenous infection through wound leak, and preexisting infection in the host tissue
    - They present with severe pain and blurred vision within a week of surgery. There is a loss of red reflex and a yellow reflex may be seen in the pupil diagnosis is established by B-scan
    - Treatment involves intravitreal antibiotics and vitrectomy
4. **Astigmatism**
    - It results from the sutures. Astigmatism is studied using serial corneal topography
    - It is corrected by the timely removal of interrupted sutures
5. **Glaucoma**
    - Glaucoma can occur in early postoperative period or late postoperative period, and is defined as a rise in IOP anytime after surgery with/without optic disc cupping
    - Causes for IOP spike in early postoperative period
        1. Preexisting glaucoma
        2. Tight sutures
        3. Viscoelstic substance
        4. Iridocyclitis
        5. Malignant glaucoma
    - . Causes of glaucoma in late postoperative period
        1. Epithelial ingrowth
        2. Fibrous downgrowth

5. Define corneal graft failure. Describe the causes, risk factors, and management of corneal graft failure. (1+1+2+1 marks)

Corneal Graft Failure

Definition

Irreversible graft edema that fails to resolve immediately after surgery

Causes

1. Poor endothelial health of the donor tissue
2. Iatrogenic damage of the donor tissue during PKP
3. High intraocular pressure
4. Infections

Risk Factors

1. Dry eye
2. Larger grafts as they are closer to the limbus
3. Corneal vascularization
4. Previous graft failure
5. Herpes simplex keratitis

Management

Regraft if the corneal edema fails to resolve.

6. Describe the risk factors, clinical features, and management of corneal graft rejection. (2+2+1 marks)

Corneal Graft Rejection

Risk Factors

1. Corneal vasulcarization
2. Young recipient age
3. Iris synechiae at graft margin
4. History of inflammatory disease
5. Prior use of antiglaucoma medications

Clinical Features

- Epithelial rejection is identified as a line of separation between healthy and rejected epithelium (Khodadaust line)
- Subepithelial rejection is recognized by infiltrates in the corneal graft
- Endothelial rejection is identified as the presence of localized stromal edema, keratic precipitates, and iritis

Management

- Topical corticosteroids
- Subconjunctival or sub-Tenon steroids
- Systemic steroids in severe rejection

7. Explain the structure and procedure of Boston keratoprosthesis. (3+2 marks)

Boston Keratoprosthesis

It is the first keratoprosthesis to get FDA approval and is the most commonly used keratoprosthesis in the world.

Structure

- It is of two types:
  - Type I: For pseudophakic and aphakic eyes with a relatively good ocular surface
  - Type II: Reserved for severe dry eyes and is placed through the lids
- The prosthesis has a front plate with an optical stem, a backplate, and a locking 'C' ring (Fig. 4.6.6)
- The front plate can be customized to suit the power of the eyes based on the axial length of the eyeball
- The prosthesis is made of PMMA

Procedure

- Following removal of the recipient cornea, the K-pro is assembled. The lens is removed
- The front plate is passed through the donor cornea and the backplate is assembled
- The backplate has 16 holes which allow the passage of nutrients to the donor cornea
- Then the donor cornea is sutured onto the recipient bed as in penetrating keratoplasty
- A bandage contact lens is placed on the K-pro

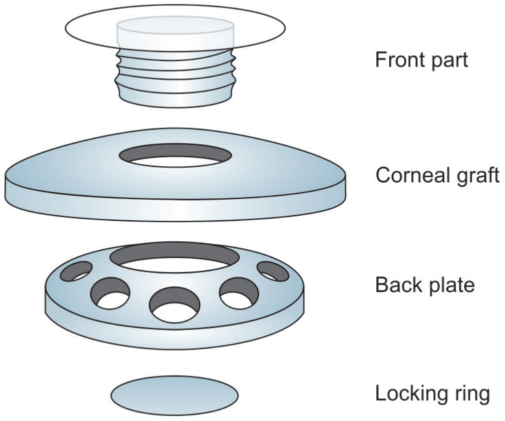


Fig. 4.6.6: Boston keratoprosthesis

8. Explain the structure of the amniotic membrane transplant. Explain its characteristics that make its use favorable in ophthalmology. (5 marks)

Structure of the Amniotic Membrane Transplant

- The amniotic membrane consists of an epithelium, basement membrane, compact layer, fibroblast layer, and an intermediate layer (Fig. 4.6.7)
- It is the inner avascular layer that is in contact with the amniotic fluid
- These layers are rich in collagen type I, III, IV, fibronectin, and proteoglycans

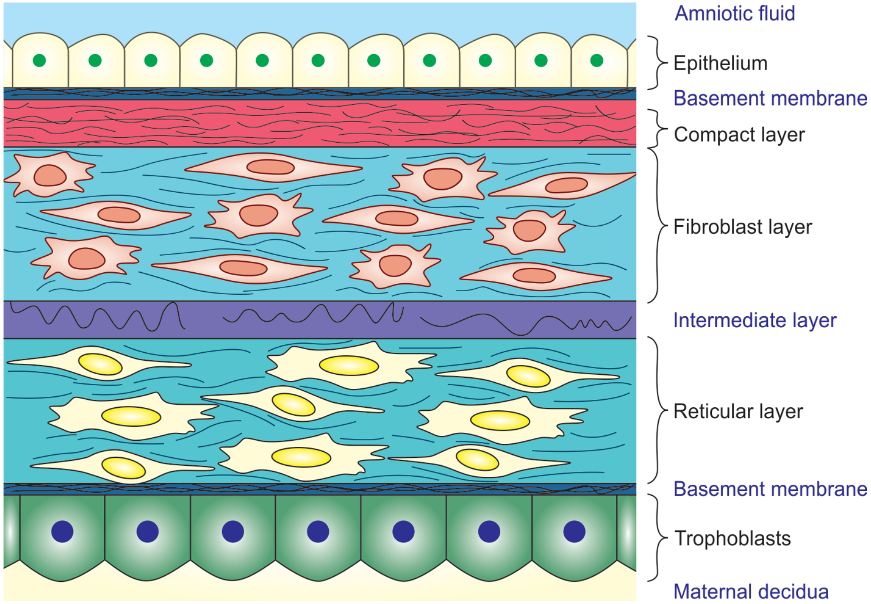


Fig. 4.6.7: Layers of amniotic membrane

Features of the Amniotic Membrane that Make it Suitable for Use in Ocular Surface Disorders

- It acts as a substrate for the rapid growth and development of corneal and conjunctival epithelial cells
- It also acts as a bandage contact lens when placed on an epithelial defect and provides adequate hydration to the epithelium
- It acts as a physical barrier from the moving eyelids and protects the ocular surface
- The stromal matrix has anti-inflammatory properties and decreases neovascularization and scarring
- It is devoid of HLA proteins and therefore there is no fear of rejection
- It acts as a barrier and keeps raw surfaces apart and thereby prevents the formation of adhesions

9. Describe the indications of amniotic membrane transplantation and explain the procedure. (5 marks)

Amniotic Membrane Transplantation

Indications

1. **Conjunctival surface reconstruction**
    - Following removal of large conjunctival lesions like pterygium, squamous cell carcinoma, etc. It has a high success rate in recurrent pterygia
    - Reconstruction following removal of symblepharon due to Stevens–Johnson syndrome, ocular cicatricial pemphigoid

2. **Corneoscleral surface reconstruction**
   - Persistent epithelial defects
   - Nonhealing corneal ulcers (both infectious and noninfectious)
   - Exposure keratopathy
   - Neurotrophic keratopathy
   - Immune-mediated stromal thinning: in this, the amniotic membrane can be used to 'fill' the defect
   - Treatment of corneal microperforations and descemetoceles
   - Use as a bandage contact lens for pseudophakic bullous keratopathy
   - Treatment of scleral melt and perforations
   - Acute stage of chemical injury to help in rapid epithelialization of the cornea

Procedure

- The amniotic membrane is stored with the epithelial surface facing away from the nitrocellulose paper
- After procurement, the membrane is adjusted to the necessary size and shape and is sutured onto the surface with a basement membrane facing up
- The stroma should be in contact with the ocular surface
- This important for proper adherence to the surface and helps in epithelial migration over it
- It is sutured onto the conjunctiva or cornea with 6-0 vicryl or 10-0 nylon
- It can be placed as an inlay or an overlay. A bandage contact lens is placed
- Postoperative antibiotics and steroids are instilled till inflammation is controlled

10. Enumerate four sites at which stem cells are present in the eye. Enumerate three causes each for unilateral and bilateral limbal stem cell deficiency. (2 + 3 marks)

Sites at Which Stem Cells are Present in the Eye

Stem cells are cells with a potential of self-renewal and producing an ongoing population of identical daughter cells of unrestricted proliferation potential.

1. Stem cells mostly reside in the limbus and are clinically seen as palisades of Vogt in the superior and inferior limbus. Loss of stem cells causes impaired healing of the corneal and conjunctival epithelium.
2. Conjunctival fornix
3. Orbital adipose tissue
4. The basal layer of skin of eyelids

Causes of Stem Cell Deficiency

Unilateral	**Bilateral**
Inflammatory • Chemical injury • Heretic infections Traumatic/Iatrogenic • Prior ocular surgery • Cryotherapy • Abuse of preservatives in medications • Contact lens-induced Tumors • Pterygium • Ocular surface squamous neoplasia	Congenital • Aniridia • Xeroderma pigmentosum • Turner syndrome Inflammatory • Stevens–Johnson syndrome • Ocular cicatricial pemphigoid • Chronic ocular allergy • Chemical burns

11. Describe the procedure of limbal stem cell transplant. (5 marks)

- The main goal of a limbal stem cell transplant is to restore the normal milieu of the ocular surface
- Limbal stem cell transplant is indicated in total limbal stem cell deficiency
- Limbal cell transplant can be autograft (same person, other eyes), allograft from a cadaver, allograft from a living-related individual, and living nonrelated
- Also, it can be classified as conjunctival, limbal, and keratolimbal transplant

Graft	Conjunctival	Limbal	Keratolimbal
Autograft	Conjunctival autograft	Conjunctival limbal autograft	Keratolimbal autograft
Allograft cadaveric	Cadaveric conjunctival autograft	Cadaveric conjunctival limbal autograft	Cadaveric keratolimbal autograft
Allograft living related (sibling or parent)	Living related conjunctival autograft	Living related Conjunctival limbal autograft	—
Allograft living nonrelated	Living nonrelated Conjunctival autograft	Living nonrelated Conjunctival limbal autograft	—

Method of Conjunctival Graft

A part of the conjunctiva is harvested and sutured to cover the defect.

Method of Conjunctival Limbal Graft

- In this technique, which remains the treatment of choice for unilateral injuries, two large free grafts, each spanning from 5 to 7 mm of limbal arc length, that is 240°, are harvested from the normal eye and transplanted to the diseased eye
- Systemic immunosuppression is required for the survival of the graft

Method of Keratolimbal Graft

- In this technique, the corneoscleral rim of tissue from cadaveric eyes is removed and sutured to the recipient bed
- This procedure is reserved for bilateral total limbal stem cell deficiency. This is supported by long-term immunosuppression

Cultivated Stem Cell Transfer

A small biopsy (2 mm × 2 mm) from the limbus from the donor's eye is taken and cultivated. These cells are transferred with the help of an amniotic membrane.

Simple Limbal Epithelial Stem Cell Transfer

- Similar to the above procedure, a small biopsy of limbal tissue is harvested from the donor's eyes and small pieces are created
- They are placed on the ocular surface of the diseased eye and allowed to expand in vivo with the help of an amniotic membrane

SHORT ANSWERS

1. Dr Harminder Singh Dua is credited for discovering a new layer in the cornea. Explain its role in keratoplasty.

Or

Explain the role of Dua's layer in keratoplasty. (3 marks)

- Dua's layer is the fourth layer of the cornea discovered by Dr Harminder Singh Dua in 2013. It marks the posterior limit of the stroma (Fig. 4.6.8)
- It is an acellular layer that is sandwiched between the stroma and the Descemet's membrane
- This layer provides a zone of separation during deep anterior lamellar keratoplasty
- As it is layer is tougher than the descemet membrane, it provides more stability following lamellar resection of the anterior cornea
- Even in the donor cornea, the posterior lamella consisting of the endothelium, DM, and the Dua's layer was thicker and stronger with lesser incidences of cracks. This improved the outcomes of endothelial keratoplasty

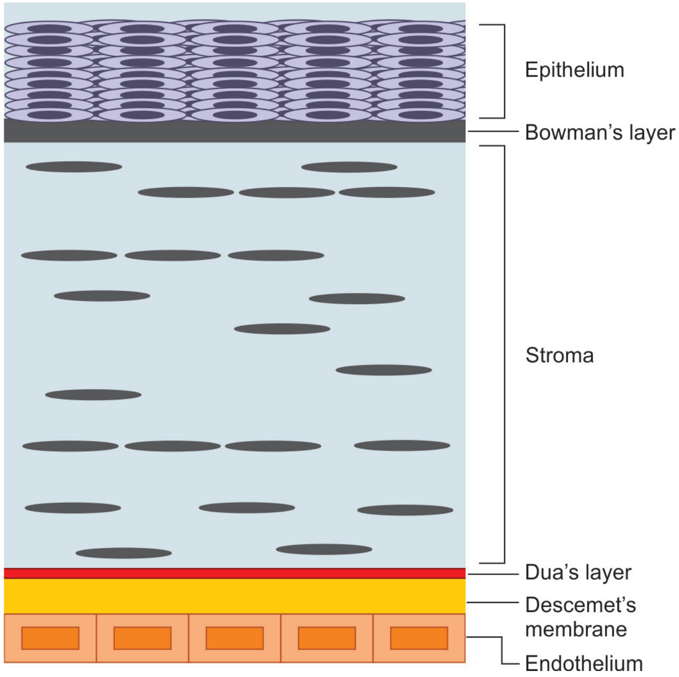


Fig. 4.6.8: Layers of the cornea

2. Explain the advantages of lamellar keratoplasty over penetrating keratoplasty. (3 marks)

- It is an extraocular procedure: The globe is not entered
- As a result, complications like wound leak, shallow anterior chamber, endophthalmitis, development of cataract, and glaucoma are absent
- Faster recovery
- Lower rates of rejection as the endothelium are not interfered with
- Lesser degrees of astigmatism in lamellar keratoplasty

3. Enumerate three corneal dystrophies that require keratoplasty. (3 marks)

1. Congenital hereditary endothelial dystrophy
2. Granular stromal dystrophy
3. Macular corneal dystrophy

4. Enumerate three conditions that are associated with poor outcomes following keratoplasty. (3 marks)

1. Limbal stem cell deficiency
2. Presence of vascularization
3. History of herpetic keratitis

5. Contrast between corneal allograft rejection and graft failure. (3 marks)

Feature	Graft failure	Graft rejection
Mechanism	Low endothelial count	Immunologically mediated
Time of presentation	Immediate postoperative day	Rarely before 2 weeks
Clinical features	Diffuse corneal edema	Presence of Khodadaust lines Stromal infiltrates KPs, iritis, and localized corneal edema
Response to steroids	Poor	Good

6. Mr H has left-sided leukomatous opacity in the central 6 mm of the cornea. He is advised keratoplasty. But the patient is apprehensive about graft rejection. He thinks the risk of rejection of corneal graft is high. Is this true? Justify your answer. (1+2 marks)

- No, this statement is false as the rate of rejection of a corneal graft is much lesser than other organ transplants
- Unlike other tissues in the body, a corneal transplant can be performed without HLA typing and between any two individuals
- The factors that contribute to this are
  - The cornea is an avascular tissue. As a result, the amount of immune elements that are delivered to the cornea is limited
  - The absence of lymphatics in the cornea prevents the entry of antigens from the cornea to lymph nodes
  - Low expression of MHC antigens
  - A low quantity of complement factors and MHC class proteins

7. Enumerate three indications for keratoprosthesis. (3 marks)

1. Vascularized cornea
2. Ocular surface inflammation as seen in Stevens–Johnson syndrome, ocular cicatricial pemphigoid
3. Limbal stem cell deficiency

COMPETENCY

OP4.7 ENUMERATE THE INDICATIONS AND DESCRIBE THE METHODS OF TARSORRHAPHY

SHORT ESSAYS

1. Enumerate four indications and explain different methods of tarsorrhaphy.

(2 + 3 marks)

Tarsorrhaphy

It is a procedure of creating temporary or permanent adhesions between a part of the upper and the lower eyelids.

Indications

The main intention is to protect the ocular surface from dryness

1. **Facial palsy**
   - This is the most common indication of tarsorrhaphy
   - Facial palsy with a high risk of exposure keratopathy due to poor Bell's phenomenon is a good candidate for tarsorrhaphy
   - A minimal amount of lagophthalmos can be managed with conservative therapy
2. **Comatose patients**
   - Tarsorrhaphy is indicated in these patients as the cornea may be exposed due to their inability to close the lids
3. **Extreme proptosis**
   - When there is extreme proptosis, the lids are unable to mechanically close the eyes due to the position of the globe
   - In such cases, a temporary tarsorrhaphy is planned
4. **Cicatrization of the skin of the lids**
   - Severe scarring may limit the functionality of the lids and increase the risk of exposure keratopathy
5. **To assist in the healing of a corneal ulcer**
   - This is a rare indication and not very commonly practiced
6. **To assist in the healing of skin grafts following oculoplastic surgeries**
   - Rotation flaps and grafts of lids need time to heal and thus need to be stationary. So, in these cases, a temporary complete tarsorrhaphy is performed

Methods of Tarsorrhaphy

- It can be classified as temporary or permanent based on the duration for which the procedure is desired
  1. **Temporary**
     - It is performed by approximating the lid at the margins with non-absorbable sutures
     - The sutures are removed when the underlying condition is corrected
  2. **Permanent**
     - It is performed by creating raw areas at the intermarginal strips of both upper and lower lids, and approximating the lids with the help of sutures (Fig. 4.7.1)

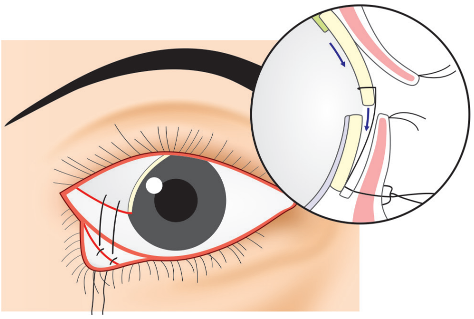


Fig. 4.7.1: Method of permanent tarsorrhaphy

  - It can also be performed by causing permanent adhesions by overlapping the lids after excising a triangular flap of skin and orbicularis from the lower lid and corresponding triangular tarsoconjunctival flap from the upper lid
- Tarsorrhaphy can also be classified as lateral and paramedian (Figs 4.7.2 and 4.7.3) based on the position of the lids that have been sutured

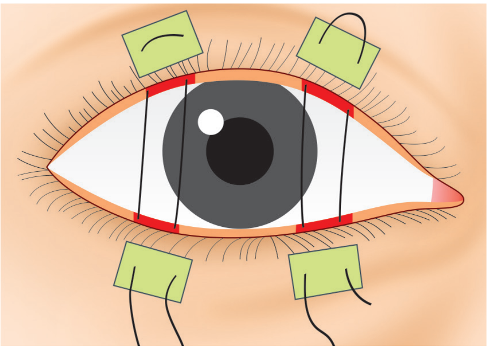


Fig. 4.7.2: Paramedian tarsorrhaphy

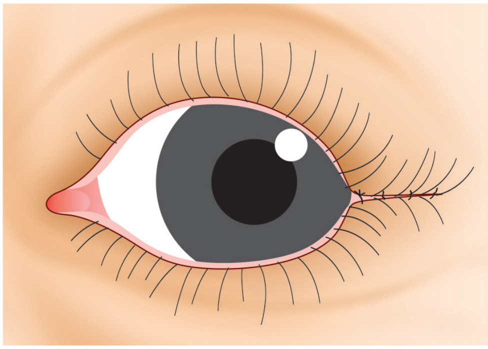


Fig. 4.7.3: Lateral tarsorrhaphy

2. Describe the causes of exposure keratopathy. Describe the management of the same. (2+3 marks)

Exposure Keratopathy

Causes

1. **Facial palsy**
   - Facial palsy can be temporary as in Bell's palsy or permanent as in a malignant parotid tumor following resection
   - When associated with poor Bell's phenomenon, the risk of exposure increases
2. **Proptosis**
   - Severe axial or eccentric proptosis may hinder adequate lid closure and cause exposure keratopathy
3. **Inability to close eyelids due to mechanical causes**
   - It may be seen due to cicatrizing changes in the skin of the lids following various inflammatory conditions
4. **Comatose patients**

Management

- Prevention of the development of exposure keratopathy can be done using taping of the eyelids during sleep and use of lubricants
- Once exposure develops, topical antibiotics and lubricants are used along with gel formulations to improve healing. Bandage contact lenses can also be used. Tarsorrhaphy (temporary or permanent) may be performed to control the healing of the exposure keratopathy
- Gold weights can be implanted within the lids to bring the lids down (traditional procedure)
- Treatment of the underlying cause

COMPETENCY

OP4.8 DEMONSTRATE TECHNIQUE OF REMOVAL OF FOREIGN BODY IN THE CORNEA IN A SIMULATED ENVIRONMENT

SHORT ESSAYS

1. Enumerate the types of corneal foreign body and their effects. (2+3 marks)

Types of Common Corneal Foreign Bodies

1. Dust while driving
2. Wood while sawing
3. Metallic foreign bodies while hammering without protective eyewear
4. Glass particles (due to car crash)
5. Adhesives like cyanoacrylate
6. Vegetative matters
7. Insects and their wings
8. Caterpillar hair

Effects of Corneal Foreign Bodies

1. Abrasion on the cornea
2. Keratitis
3. Formation of rust ring in cases of iron foreign body
4. Penetration of the cornea and rarely perforation and open globe injury with intraocular foreign body

2. Describe the clinical features and management of corneal foreign bodies. (2+3 marks)

Corneal Foreign Bodies

Clinical Features

- The presentation is typical: Severe pain and photophobia with a history of entry of foreign body are usually present
- There is conjunctival congestion
- If the foreign body is metallic, after 4–6 hours, there is the formation of a rust ring around the foreign body. However, they enter with high velocity and get sterilized due to heat
- Soil and vegetative matter are more likely to cause infection and keratitis

Management

- Anesthetize the eye with proparacaine. The patient is seated at the slit-lamp
- Localize with a light beam and instruct the patient to be very still. The foreign body is removed (along with the rust ring if present) using a 26 gauge needle or a foreign body spud
- Removal of the foreign body under topical anesthesia followed by topical antibiotics
- If there is no inflammation topical antibiotics are prophylactically given (Ciprofloxacin or ofloxacin QID for 2–3 days)
- If there is inflammation, then it should be treated with cycloplegics and antibiotics under observation (like a corneal ulcer)

COMPETENCY

OP4.9 DESCRIBE AND DISCUSS THE IMPORTANCE AND PROTOCOLS INVOLVED IN EYE DONATION AND EYE BANKING

SHORT ESSAYS

1. Define eye banking. Describe the protocols for eye retrieval, storage, and procurement of cornea. (1+4 marks)

Definition of Eye Banking

A system that encompasses eye retrieval, grading, storage, and distribution to trained surgeons for corneal transplantation

Eye Retrieval

- It is done by a trained medical professional associated with an eye bank
- It is done after confirming the death of the individual
- Retrieval is done by enucleation or corneoscleral rim procedure
- The first step in this procedure to meet the kin of the deceased and collect the death certificate and consent for retrieval
- A general survey of the body is performed and any needle marks, ulcers, gangrene must be looked for
- A social history of the diseased must be obtained and various lists of contraindications must be ruled out
- A sample of blood from the donor is drawn for HIV, HBsAg and HCV, HTLV-1 and 2, and syphilis

Eye Storage

- The enucleated eye or the corneoscleral rim is stored in a moist chamber and refrigerated at 4°C. within the next 24 hours, the eye should be evaluated using a slit lamp.
- Grading is done based on the epithelium, stroma, Descemet's membrane, and endothelial health. Also, specular microscopy is performed

Parameter	Grade 1 (Excellent)	Grade 2 (Very good)	Grade 3 (Good)	Grade 4 (Fair)	Grade 5 (Poor)
Epithelial defects and haze	None	Slight	Obvious	—	—
Stromal clarity	Crystal clear	Clear	Slight cloudiness	Moderate cloudiness	Severe cloudiness
Arcus senilis	None	Slight	Moderate (<2.5 mm)	Heavy (2.5–4 mm)	Very heavy (>4 mm)
Descemet's membrane	No folds	Few shallow folds	Numerous shallow folds	Numerous deep folds	Marked deep folds
Endothelium	No defect	No defect	Few vacuolated cells	Moderate guttate	Marked guttate

- The container is labeled as regards the details of the eye bank, time and date of death, type of preservation, etc.

Procurement of the Donor Cornea

- The recipient has to register through an eye bank
- When there is an availability of donor cornea, the eye bank takes upon itself the job of procuring the donor eye to the recipient

2. Name the methods of donor cornea storage along with their properties. (5 marks)

Period	Types	Temperature	Time limit	Constituents
Short-term	Moist chamber	4 degrees	24 hours	—
	McCarey Kauffmann	4 degrees	2–3 days	Dextran, HEPES buffer, gentamicin, and phenol red
Intermediate-term	K-sol Dexol Optisol	2–6 degrees	7–10 days	MK medium + chondroitin sulfate
	Optisol GS	2–6 degrees	14 days	Add gentamicin and streptomycin
Long-term	Organ culture	31–37 degrees	35 days	Minimal essential medium supplemented with fetal calf serum
Very long-term	Cryopreservation	Freezing and vitrification	1 year	Glycerol

SHORT ANSWER

1. Enumerate six contraindications for corneal donation. (3 marks)

1. Death due to an unknown cause
2. Death due to rabies, Cruzefeld Jacob disease, septicemia
3. Presence of HIV, HBsAg, SSPE
4. Congenital rubella syndrome
5. Active ocular inflammation
6. Homosexual, IV drug abuse, tattooing

COMPETENCY

OP4.10 COUNSEL PATIENTS AND FAMILY ABOUT EYE DONATION IN A SIMULATED ENVIRONMENT

SHORT ANSWER

1. Describe the functions of an Eye Bank. (3 marks)

Functions of an Eye Bank

1. The increasing rate of eye donation by creating and increasing awareness about eye donation to the general public
2. Registration of increasing number pledgers for eye donation
3. Retrieval of the donor's eyes from the deceased and processing them
4. Preservation of the tissue for short-, intermediate-, long-, or very long-term
5. Distribution of the donor tissues to the corneal surgeons at the time of need
6. Conducting research activities for further improvements of the preservation techniques and utilization of the other components of the eye (example sclera)

LONG ESSAY (MISCELLANEOUS)

1. A corneal surgeon's paraphernalia includes a set of investigations.

A. Enumerate three special imaging techniques for studying various parameters of the cornea. (3 marks)

B. Describe the utility of corneal topography. (3 marks)

C. Describe the utility of specular microscopy. (3 marks)

D. State the use of a corneal esthesiometer. (1 mark)

A. Imaging Techniques for Studying the Cornea

1. **Pachymeter:** To measure the thickness of the cornea
2. **Specular microscopy:** To study the endothelium of the cornea
3. **Corneal topography:** To study the surface, curvature, dioptric power, and thickness of the cornea

B. Corneal Topography

- Corneal topography is the study of the surface of the cornea and has evolved from using Placido disc to Pentacam
- The principle of topography is using Placido-disc mires to reflect light off the anterior corneal surface and reflected light is studied using computer-based imaging.

Clinical Uses

1. **Refractive surgery**
   - Corneal topography is a must-tool for refractive surgeries
   - Irregular corneas with thinning are poor candidates for refractive surgery
   - Also, it allows to diagnose Formefruste keratoconus which is a contraindication for refractive surgeries
2. **Diagnosis and follow-up of ectasia-related disorders**
   - Keratoconus and post-LASIK ectasia are frequently encountered ectatic conditions that are diagnosed using corneal topography
   - Keratoconus is characterized by inferior steepening and thinning. Early keratoconus is picked up on pentacam and posterior float elevation is diagnostic
3. **Evaluation of astigmatism**
   - Corneal topography is used to diagnose and study the effects of various lesions like pterygium, corneal incisions, on corneal astigmatism
   - Also, it is useful before planning an astigmatic keratotomy

C. Specular Microscopy

This method allows evaluation and imaging of the corneal endothelium

Clinical Uses

1. **Diagnosis and follow-up of corneal dystrophies**

Specular microscopy forms an integral part of the diagnosis of endothelial dystrophies, like Fuch's endothelial dystrophy, posterior polymorphous dystrophy, and ICE syndromes	Presence of Descemet excrescences like guttae Fuch's endothelial dystrophy Loss of hexagonality of endothelium

Contd.

Posterior polymorphous dystrophy	Donut-shaped vesicles with clearly defined black margins are seen anterior to the endothelium
ICE syndromes	Intracellular dark areas are seen within the endothelium Reversal sign: Black areas with white margins

2. **Evaluation before an intraocular surgery**
    - There is a risk of corneal edema when the endothelial count is <700 cells/mm^2, co-efficient of variance >40%, or if the percentage of hexagonality <50%
    - Preoperative assessment allows predicting the risk of corneal edema following surgery
3. **Donor cornea evaluation**
    - Assessment of endothelium to grade the donor corneal button before transplantation

D. Corneal esthesiometer

Used to measure corneal sensation

SHORT ESSAYS

1. Enumerate four congenital corneal anomalies. Describe the clinical features of megalocornea. (2+3 marks)

Congenital Corneal Anomalies

a. Megalocornea
b. Microcornea
c. Keratoglobus
d. Cornea plana

Clinical Features of Megalocornea

- It is a rare, nonprogressive hereditary condition
- X-linked recessive, hence affects males
- May be isolated or associated with systemic component
- Systemic associations are:
    - Neuhauser syndrome: Megalocornea + hypotony + mental retardation
    - Down syndrome
    - Albinism

Symptoms

- Discovered at birth
- May present with refractive error

Signs

- Adult cornea >13 mm of horizontal diameter is characteristic
- Deep anterior chamber
- High myopia
- May be associated with subluxation of the lens due to stretching
- Predisposed to glaucoma

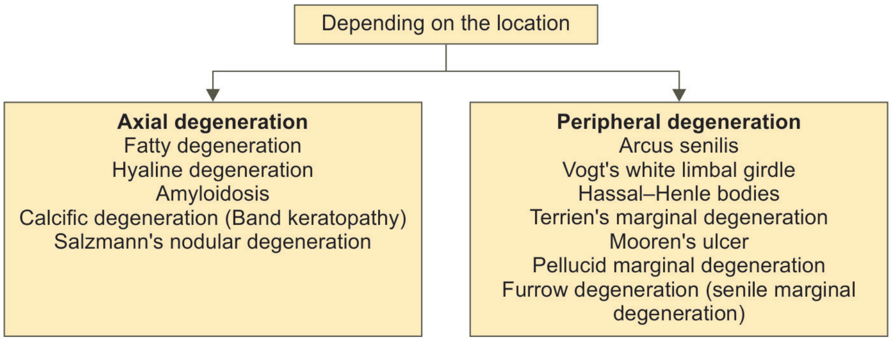
2. Enumerate various congenital corneal anomalies and mention one specific feature of each. (5 marks)

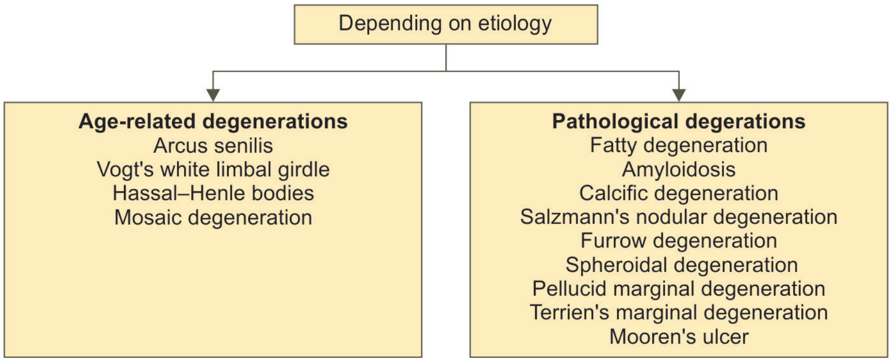
Congenital Corneal Anomalies Include

Anomalies of shape	
Megalocornea	Increased horizontal diameter of the cornea (>11.7 mm)
Microcornea	Decreased horizontal diameter of the cornea (<10 mm)
Cornea plana	An autosomal dominant condition is characterized by the flat cornea
Congenital corneal absence	It may be associated with cryptophthalmos
Congenital anterior staphyloma or keratoglobus	Ectasia of the cornea leading to the formation of a globe-like cornea
Anomalies of clarity	
Anterior embryotoxon	Apparent widening of the superior limbal transition between the sclera and the cornea
Posterior embryotoxon	Thickening and anterior displacement of the Schwalbe's line Seen in association with Axenfeld anomaly
Corneal keloids	They appear as white glistening lesions involving the whole or part of the cornea
Dermoids	Usually seen in the inferotemporal aspect of the limbus Usually round, pinkish-white, dome-shaped, with other skin appendages Associated with Goldenhar syndrome
Sclerocornea	Nonprogressive, noninflammatory, sclera-like opacification of the cornea It may be associated with glaucoma
Congenital hereditary endothelial dystrophy	Diffuse bilateral corneal edema and clouding without any rise in intraocular pressure

3. Classify corneal degenerations. Describe the clinical features of band-shaped keratopathy. (3+2 marks)

Classification of Corneal Degenerations


Depending on the location
Axial degeneration
Fatty degeneration
Hyaline degeneration
Amyloidosis
Calcific degeneration (Band keratopathy)
Salzmann's nodular degeneration
Peripheral degeneration
Arcus senilis
Vogt's white limbal girdle
Hassal–Henle bodies
Terrien's marginal degeneration
Mooren's ulcer
Pellucid marginal degeneration
Furrow degeneration (senile marginal degeneration)


Depending on etiology
Age-related degenerations
Arcus senilis
Vogt's white limbal girdle
Hassal–Henle bodies
Mosaic degeneration
Pathological degerations
Fatty degeneration
Amyloidosis
Calcific degeneration
Salzmann's nodular degeneration
Furrow degeneration
Spheroidal degeneration
Pellucid marginal degeneration
Terrien's marginal degeneration
Mooren's ulcer

Clinical Features of Band-shaped Keratopathy

- It is seen as whitish powder deposits, starting at the nasal and temporal sides of the cornea with a clear zone between it and the limbus
- These later extend into the central cornea
- They are seen at the level of the Bowman's membrane
- It is also called swiss cheese appearance

4. Classify corneal dystrophies. Elaborate on the clinical features of Map-dot-fingerprint dystrophy. (3+2 marks)

Classification of Corneal Dystrophies

1. **Epithelial**
    i. Map-dot-fingerprint dystrophy
    ii. Reis-Buckler's dystrophy
    iii. Meesman's dystrophy
    iv. Recurrent corneal erosion syndrome
    v. Stocker-Holt dystrophy
2. **Stromal**
    i. Granular dystrophy
    ii. Lattice dystrophy
    iii. Macular dystrophy
    iv. Crystalline dystrophy
3. **Endothelial**
    i. Fuch's endothelial dystrophy
    ii. Congenital hereditary endothelial dystrophy
    iii. Posterior polymorphous dystrophy

Clinical Features of Map-dot-fingerprint Dystrophy

- It is common dystrophy, seen usually in adults with sporadic distribution occurring due to loss of hemidesmosome between the basement membrane and the epithelium
- These individuals present with dot-like or fingerprint pattern-like opacities in the anterior part of the cornea
- As a result, the cornea is very susceptible to erosions
- Symptoms are mild pain and ocular irritation, and on examination, one can find multiple dot-like or map or fingerprint-like opacities, and due to this varied appearance, it is often misdiagnosed

5. Enumerate any two endothelial dystrophies. Describe the clinical features and management of any one of them. (2+3 marks)

Endothelial Dystrophies

1. Fuch's endothelial dystrophy
2. Posterior polymorphous dystrophy

Fuch's Endothelial Dystrophy

Clinical Features

- Presents at the age of 50–60 years
- Bilateral decrease in the endothelial count

Symptoms

- In the early stages, there are no symptoms
- Slowly, patients may notice mild blurring of vision and pain in the eye, more marked in the mornings on awakening
- These symptoms become more marked with the progression of the disease

Signs

- Corneal guttata (excrescences) of Descemet's membrane
- Stromal edema due to endothelial decompensation
- Epithelial edema and bullous keratopathy
- Corneal scarring

Management

- Bandage contact lens
- Hypertonic saline drops
- Endothelial keratoplasty (Descemet's membrane endothelial keratoplasty and Descemet's stripping automated endothelial keratoplasty)

6. A 17-year-old boy presents with a diminished vision for distance. On performing retinoscopy, there is a scissoring reflex. The manifest refraction is –5.0D sphere –4.0 cyl at 60° in the right eye, and –6.5D sphere –5.0 cyl at 110° in the left eye.

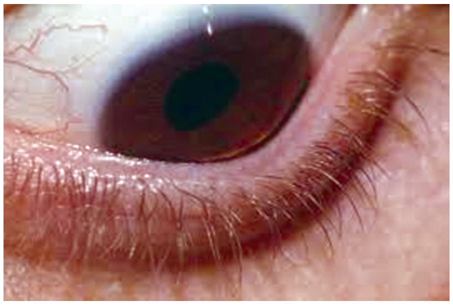
A. What is the most probable diagnosis? (1 mark)

B. Describe the clinical features of the same. (4 marks)

A. Most Probable Diagnosis

Keratoconus

B. Clinical Features of Keratoconus



Symptoms

- The affection is bilateral but is asymmetric
- The symptoms are mainly visual with blurring and distortion being the most common
- They also present with frequent changes of glasses, which they are usually unhappy with

- It is seen more in teenage boys, which progresses until the late twenties or early thirties and then burns out

Signs

- Window reflex is distorted
- On distant direct ophthalmoscopy, oil droplet reflex is seen
- Scissoring reflex is seen on retinoscopy
- On shining light on the temporal side of the cornea, a shadow of the cone is formed on the nasal conjunctiva—Rizzuti sign
- On slit-lamp examination, the thinning and ectasia of the central cornea with iron deposition at the base of the cone—Fleischer's ring
- Very fine, vertical, deep stromal 'Vogt's striae are seen and they disappear with external pressure on the globe
- Keratoscopy mires show inferonasal crowding
- Munson sign is seen in severe keratoconus: Bulging of the lower lid while downgaze

SHORT ANSWERS

1. Enumerate stromal corneal dystrophies. Describe the clinical features of each. (3 marks)

Stromal Corneal Dystrophies

1. Granular dystrophy
2. Macular dystrophy
3. Lattice dystrophy

Clinical Features of Each

Name of dystrophy	Clinical features
Granular dystrophy	Small, white granular opacities in the stroma of the central cornea, surrounding corneal haze, limbus is spared
Macular dystrophy	Dense, gray opacities in the central cornea presents in early childhood
Lattice dystrophy	Spider-like branching opacities in the stroma and form a lattice, presents within 20 years of age

2. Describe the corneal manifestations of any three metabolic disorders. (3 marks)

Metabolic disorder	Corneal manifestations
Cystinosis	• Corneal and conjunctival deposits of cystine are seen as bright refractile golden crystals • They present with severe photophobia and lacrimation
Wilson's disease	• Keyser–Fleischer ring (green-yellow ring-shaped opacity in the peripheral part of Descemet's membrane) • It is of diagnostic value • Disappears with treatment • May develop anterior capsular 'sunflower cataract'
Fabry's disease	• Vortex keratopathy (whorl like opacities in the cornea) • These ate visually insignificant

3. Describe the etiopathology of Band-shaped keratopathy. (3 marks)

- It is a pathological degeneration characterized by the deposition of calcium salts (calcium hydroxyapatite) at the level of deep epithelium and Bowman's membrane
- It occurs as a result of chronic inflammation in the eye

Etiopathology

- Calcium salts get precipitated when pH increases due to chronic inflammation and an increase in calcium concentration
- It occurs in the interpalpebral area due to loss of solvents by evaporation and loss of CO_2 and thus increasing the pH
- Chronic inflammation: Still's disease in children, any type of chronic uveitis, interstitial keratitis, phthisis bulbi
- Hypercalcemia: Hyperparathyroidism, hypervitaminosis D, milk alkali syndrome, sarcoidosis
- Elevated serum phosphorus with normal calcium (end-stage renal disease)
- Chronic exposure to mercury vapors or mercurial preservatives in ophthalmic solutions
- Silicone oil implantation in the aphakic eye (if it enters the anterior chamber)
- Hereditary causes

4. Compare and contrast corneal dystrophies and degenerations. (3 marks)

Features	**Degenerations**	**Dystrophies**
Age at presentation	Presents later in life, is age-related	Hereditary, presents early in life
Laterality	Asymmetric, usually bilateral	Always symmetric, bilateral as a rule
Pattern of opacification	Opacification is usually in the peripheral cornea	Opacification is usually central
Progression	Can be fast or slowly progressive	Usually slowly progressive

5. Explain the principles and enumerate three uses of specular microscopy. (2+1=3 marks)

- This method allows evaluation and imaging of the corneal endothelium
- Principle: As light passes from a medium of higher refractive index to a medium of lower refractive index, most of the light gets reflected off the interface. Here, the interface is formed by the corneal endothelium
- By specular microscopy, one can study the number of endothelium/mm^2, the shape of the endothelium, presence of abnormal structures (e.g. guttae), and the software allows calculation of various parameters like standard deviation and coefficient of variance

Clinical Uses

1. Diagnosis and follow-up of corneal dystrophies
2. Evaluation before an intraocular surgery
3. Donor cornea evaluation

6. Describe the ocular features of Wilson's disease. (3 marks)

- Wilson's disease or hepatolenticular degeneration develops due to decreased blood ceruloplasmin levels
- The ocular features include
  - Keyser–Fleischer ring (green-yellow ring-shaped opacity in the peripheral part of Descemet membrane)
  - It is of diagnostic value
  - Disappears with treatment
  - May develop anterior capsular 'sunflower cataract'

7. Grade keratoconus based on keratometry. (3 marks)

Grading of Keratoconus

Based on the keratometer reading

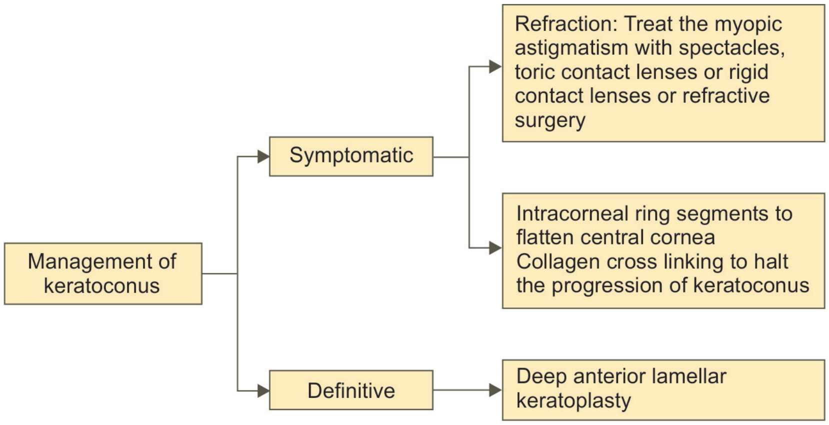
1. Mild: <45D in both meridian
2. Moderate: 45–52D in both meridian
3. Severe: >52D in both meridian
4. Advanced: >62D in both meridian

8. Describe the complications of keratoconus. (3 marks)

Complications of Keratoconus

1. Acute hydrops is sudden localized corneal edema due to a break in the Descemet's membrane due to extreme stretching and entry of aqueous into the stroma
2. It is an extremely painful condition
3. It is a sort of 'speed breaker' to the disease progression as the scarring induced bythis halts further progression. However, the visual acuity decreases as a result of scarring

9. Summarize the management of keratoconus. (3 marks)


Management of keratoconus
Symptomatic
Definitive
Refraction: Treat the myopic astigmatism with spectacles, toric contact lenses or rigid contact lenses or refractive surgery
Intracorneal ring segments to flatten central cornea
Collagen cross linking to halt the progression of keratoconus
Deep anterior lamellar keratoplasty

10. Explain the following concerning keratoconus:

A. Rizutti sign (1 mark)

B. Munson's sign (1 mark)

C. Charleaux sign (1 mark)

A. Rizzuti's Sign

- On shining light on the temporal side of the cornea, a shadow of the cone is formed on the nasal conjunctiva

B. Munson's Sign

- It is a sign of severe keratoconus
- There is a bulging of the lower lid while downgaze

C. Charleaux Sign

- It is a finding on distant direct ophthalmoscopy
- There is an appearance of a dark reflex in the area of the cone seen through a dilated pupil

11. Describe corneal topography findings in keratoconus. (3 marks)

- Corneal topography is the study of the corneal surface and its curvature
- The Rabinowitz criteria are used to diagnose keratoconus
  - K> 47.2D
  - Inferior vs. superior corneal diopteric asymmetry value > +1.4D
  - KISA% >60% is suggestive of keratoconus
- Another feature is the elevation of the posterior float >47 µm

12. Describe the corneal manifestations of cystinosis. (3 marks)

- Cystinosis is an autosomal recessive, lysosomal storage disorder characterized by the deposition of cystine (an amino protein) in various tissues of the body
- It occurs due to a defective enzyme, cystinosin which is a transporter protein responsible for transporting cystine out of the lysosome
- As a result, cysteine accumulates within the lysosome and damages the cells

Symptoms

- The patients present with severe photophobia and lacrimation
- In chronic cases, there may be visual impairment

Signs

- Severe photophobia
- Ocular manifestations include corneal and conjunctival deposits of cystine are seen as bright refractile golden crystals
- There may epithelial erosions

13. Describe the corneal manifestations of Fuch's endothelial dystrophy. (3 marks)

- Fuch's endothelial dystrophy is an autosomal dominant condition and presents at the age of 50–60 years, a bilateral decrease in the endothelial count
- On slit-lamp examination, the following features are seen:
  - Corneal guttata (excrescences) of Descemet's membrane
  - Stromal edema due to endothelial decompensation
  - Epithelial edema and bullous keratopathy
  - Corneal scarring

14. Describe the ocular manifestations of Axenfeld–Reiger anomaly. (3 marks)

Axenfeld–Reiger Anomaly

- An autosomal dominant condition
- Characterized by the following ocular features

- Bilateral posterior embryotoxon (anteriorly displaced, thickened Schwalbe's line)
- Iris anomalies
- Corectopia (ectopic pupil)
- Iris atrophy
- Developmental glaucoma

15. Describe the diagnostic criteria of megalocornea. (3 marks)

It is an X-linked recessive condition and thus affects males more (90%)

Horizontal diameter of the cornea	Normally	Megalocornea
At birth	10 mm	>11.7 mm (adult size at birth)
After 2 years	11.7 mm (adult size)	>13 mm

16. Describe the clinical features of arcus senilis. (3 marks)

- It is the most common bilateral corneal degeneration seen in almost all people over the age of 80 years, and in 60% of individuals aged 40–60 years
- It is called anterior gerontoxon
- No symptoms
- It is described as bluish-gray circular opacity of width 1–2 mm occurring 1 mm away from the limbus
- This clear zone separating limbus and arcus is called the limbal interval of Vogt
- It begins at the upper and lower poles, which later meet on the sides
- When detected in individuals of age less than 40 years, they should be investigated for hypercholesterolemia

17. Enumerate the ocular associations of keratoconus. (3 marks)

1. Vernal keratoconjunctivitis
2. Retinitis pigmentosa
3. Leber's congenital amaurosis

CHAPTER

5

Sclera

COMPETENCY

OP5.1 DEFINE, ENUMERATE AND DESCRIBE THE AETIOLOGY, ASSOCIATED SYSTEMIC CONDITIONS, CLINICAL FEATURES, COMPLICATIONS, INDICATIONS FOR REFERRAL AND MANAGEMENT OF EPISCLERITIS

SHORT ESSAYS

1. Enumerate four systemic conditions associated with episcleritis and outline its clinical features. (2+3 marks)

Systemic Associations of Episcleritis

1. Rheumatoid arthritis—the most common
2. Inflammatory bowel disease (Crohn's disease and ulcerative colitis)
3. Psoriatic arthritis
4. Systemic lupus erythematosus

Clinical Features of Episcleritis

Symptoms

- It is asymptomatic many a time
- Localized redness on side of the eye, often noticed by others
- Some may present with foreign body sensations

Signs

Diffuse episcleritis	Nodular episcleritis
The eye appears diffusely congested, however, one or two quadrants may be more congested	One pink or purple flat nodule may be seen, usually on the temporal aspect, surrounded by deep, red, centripetal vessels
The congestion is deeper to the conjunctiva and is dark red	The congestion is deeper to the conjunctiva and is dark red
Mild tenderness	Mild tenderness

- The rest of the anterior segment and fundus is normal.

2. Enumerate five differential diagnoses for a limbal nodule and explain their unique features. (5 marks)

Dermoid (congenital cause)	• Seen most at the inferotemporal quadrant • May encroach onto the cornea • No associated congestion • Well-rounded margins • Appears whitish pink • Associated with Goldenhar syndrome
Nodular scleritis (inflammatory)	• Associated with severe pain and purplish hue of vessels • Congestion does not blanch with phenylephrine
The limbal variant of vernal catarrh (Allergic)	• History of severe itching, the condition is bilateral • Gelatinous 1 mm diameter nodules at the limbus, with dusky congestion
Conjunctival ulcer (infectious)	• Stains with fluorescein • Painful • Responds well to scraping and antibiotics • Flares with steroids
Pinguecula (degenerative)	• Yellowish white fat-like appearing nodule • No congestion or pain

SHORT ANSWER

1. Annotate the management of episcleritis. (3 marks)

- Episcleritis is a self-limiting condition that subsides by a week
- Topical NSAIDs eye drops like Bromfenac, Nepafenac are given thrice a day for a week
- Topical corticosteroid eyedrops (like fluorometholone and loteprednol) are prescribed if NSAIDs do not seem to be helping
- Oral NSAIDs like indomethacin, ibuprofen, flurbiprofen may be needed in recurrent cases, especially if associated with systemic disorders
- Supportive treatment can be given in the form of tear film substitutes and cold compresses over the lids

COMPETENCY

OP 5.2 DEFINE, ENUMERATE AND DESCRIBE THE AETIOLOGY, ASSOCIATED SYSTEMIC CONDITIONS, CLINICAL FEATURES, COMPLICATIONS, AND INDICATIONS FOR REFERRAL AND MANAGEMENT OF SCLERITIS

LONG ESSAY

1. An 80-year-old woman with long-standing rheumatoid arthritis complains of seeing blue in the white of her eye. On examination, there is a localized area of scleral thinning without surrounding congestion.

A. State the most probable diagnosis. (1 mark)

B. Classify scleritis. (3 marks)

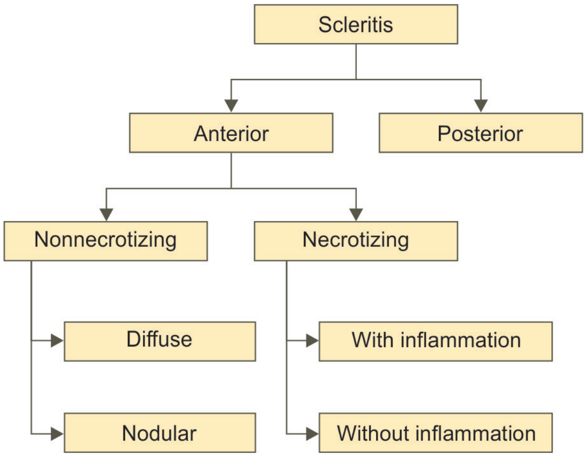
C. State one complication and elaborate on the treatment options for scleritis. (4 marks)

D. How does management in this case differ from other types of scleritis? (2 marks)

A. Most Probable Diagnosis

Non-inflammatory scleritis, also known as scleromalacia perforans, a common form of scleritis seen in rheumatoid arthritis

B. Classification of Scleritis



C. Complication

Staphyloma formation

Treatment Options

- Scleritis usually has a systemic component, and the presence of scleritis indicates a rapid progression of the disease which may prove fatal in certain systemic conditions
- The treatment for scleritis is stepwise and the algorithm is as follows
  - Oral NSAIDs
  - Oral Prednisolone 1 mg/kg for a week followed by a tapering dose of 20 mg/day for 2–3 weeks
  - Immunosuppressives like Methotrexate, Cyclophosphamide, Azathioprine if steroids do not give an adequate response
  - Concurrent topical steroids and cycloplegics and systemic proton pump inhibitors are needed

D. Comparison of Management with Other Types of Scleritis

- This patient has a necrotizing non-inflammatory type of scleritis
- The affected part of the sclera separates itself from the surrounding sclera, becomes white, and gets thinned out
- This is usually followed by a scleral melting
- Treatment in the form of systemic steroids and immunosuppressives is usually insufficient and usually requires a scleral patch graft. It is obtained from the sclera of done using donor eyes retrieved for keratoplasty
- After carefully debriding the necrotic sclera, the donor sclera is fashioned to suit the appropriate size, contour, and thickness and sutured with 8-0 vicryl. It is then covered by the conjunctiva.

SHORT ESSAYS

1. Enumerate four systemic associations of scleritis. Explain the clinical features of scleritis. (2+3 marks)

Systemic Associations of Scleritis

1. Rheumatoid arthritis
2. Wegener's granulomatosis or granulomatosis with polyangiitis
3. Systemic lupus erythematosus
4. Behcet's disease

Clinical Features of Scleritis

- It depends on the type of scleritis
- Pain and redness are the two most important presentations in anterior scleritis
- The pain is severe
    1. **Nodular scleritis**
        - A swelling, more diffuse than episcleritis may be seen on the sclera, which is reddish-blue, to begin with, but later turns purple due to scleral thinning
        - It may spread around the cornea in an annular fashion to give rise to 'annular scleritis'
    2. **Diffuse scleritis**
        - White opacities can appear surrounded by congested vessels
    3. **Necrotizing scleritis with inflammation**
        - There is necrosis due to vasculitis
        - There is scleral thinning associated with sloughing and associated uveitis
    4. **Necrotizing scleritis without inflammation: "Scleromalacia perforans"**
        - The clear thins and melts without any associated inflammation
        - A patch of ischemic sclera develops, forming a sequestrum that slowly separates from the surrounding healthy sclera and melts. It is due to obliterative arteritis
    5. **Posterior scleritis**
        - There are inflammation and thickening of the posterior part of the sclera
        - There is no associated connective tissue disease
        - The presentation is different from anterior scleritis. B scan is the diagnostic test that shows a thickened posterior sclera (T sign)
        - The presentation is different from anterior scleritis
        - They can present with:
            - Decreased visual acuity
            - Painful and restricted eye movements
            - Disc edema
            - Macular edema
            - Choroidal folds
            - Choroidal detachment
            - Exudative retinal detachment

2. Annotate the etiology and clinical features of posterior scleritis. (2+3 marks)

Posterior Scleritis

Etiology

- Posterior scleritis is inflammation of the posterior part of the sclera, i.e., behind the equator

- Systemic associations are less common than with anterior scleritis
- Rheumatoid arthritis and Wegener's granulomatosis are the most common causes
- Others are tuberculosis, syphilis

Clinical Features

- The eye is usually quiet-looking with no signs of inflammation
- Patients may complain of severe headache
- They usually present with diminution of vision associated with non-specific features such as
  - Painful and restricted eye movements
  - Disc edema
  - Macular edema
  - Choroidal folds
  - Choroidal detachment
  - Exudative retinal detachment
  - Diagnosis is based on clinical suspicion and confirmed by B-scan that shows T-sign. The 'T' is formed by fluid in Tenon's space and along with the optic nerve shadow

3. Define staphyloma. Categorize staphyloma and enumerate one cause for each type. (1+2+2 marks)

Definition of Staphyloma

Thinning or ectasia of the outer coat of the eyeball (cornea or sclera) along with the incarceration of uveal tissue.

Type of staphyloma	Anterior staphyloma	Intercalary staphyloma	Ciliary staphyloma	Equatorial staphyloma	Posterior staphyloma
Site of staphyloma	Occurs when more than 2/3rd of the cornea is compromised	Occurs in the limbal area	2–3 mm behind the limbus	At the equatorial region, where the vortex veins enter	In the posterior pole
Part of uvea incarcerated	Iris	Root of iris	Ciliary body	Choroid	Choroid
Important cause	Corneal ulcer	Perforating injury that has improperly healed	Absolute glaucoma	Scleritis	Degenerative myopia

SHORT ANSWERS

1. Elucidate the clinical features of surgically induced necrotizing scleritis. (3 marks)

Clinical Features of Surgically Induced Necrotizing Scleritis

- Surgically induced necrotizing scleritis (SINS) is a type of necrotizing anterior scleritis with inflammation
- It can present over a varied period ranging from 8 days postoperatively to up to 9 months
- It usually follows cataract surgery as it is the most commonly performed intraocular surgery.
- It can also follow trabeculectomy, pterygium excision, and retinal detachment surgeries

- Any appearance of pain that cannot be managed by NSAIDs should arouse a suspicion of SINS or endophthalmitis
- The signs of inflammation are seen at the incision or operative site associated with scleral thinning
- A positive ANA or RA is not necessary for the diagnosis of SINS. However, the presence of a positive RA/ANA factor does not rule out the diagnosis of SINS
- Treatment is in the form of systemic steroids and immunosuppressants

2. Enumerate four causes for blue sclera. (3 marks)

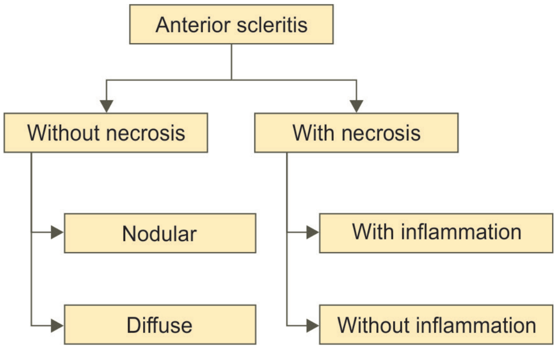
1. Pseudoxanthoma elasticum
2. Osteogenesis imperfecta
3. Weill–Marchesani syndrome
4. Marfan's syndrome
5. Buphthalmos

3. Give an indication for scleral patch graft. Classify anterior scleritis. (1+2 marks)

Indication for Scleral Patch Graft

Scleral melt due to scleromalacia perforans

Classification of Anterior Scleritis



4. Explain the terms

A. Lamina cribrosa (1 mark)
B. Lamina fusca (1 mark)
C. Tenon's fascia (1 mark)

A. Lamina Cribrosa

Sieve-like opening in the posterior sclera that allows the entry of the optic nerve.

B. Lamina Fusca

Innermost layer of the sclera that blends with the suprachoroidal space and supraciliary lamina.

C. Tenon's Fascia

It is the outer covering of the sclera that covers the whole globe from the limbus to the entry of the optic nerve.

CHAPTER

6

Iris and Anterior Chamber

COMPETENCY

OP6.1 DESCRIBE CLINICAL SIGNS OF INTRAOCULAR INFLAMMATION AND ENUMERATE THE FEATURES THAT DISTINGUISH GRANULOMATOUS FROM NONGRANULOMATOUS INFLAMMATION. IDENTIFY ACUTE IRIDOCYCLITIS FROM CHRONIC CONDITION

LONG ESSAYS

1. A 24-year-old woman presents with gradual onset of pain and redness in her right eye. on examination, there is ciliary congestion, corneal endothelium is studded with greasy mutton fat keratic precipitates in the inferior part. The pupil is sluggish, and the anterior chamber shows 2+ cells and 2+ flare.

A. What is the most probable diagnosis and what is its type? (2 marks)
B. Name three causes for this condition. (3 marks)
C. Explain the influence of this condition on intraocular pressure. (3 marks)
D. Name two posterior segment signs of this condition. (2 marks)

A. Most Probable Diagnosis

- Iridocyclitis (anterior uveitis)
- It is of 'granulomatous' type

B. Causes for Granulomatous Iridocyclitis

1. Tuberculosis
2. Sarcoidosis
3. Vogt–Koyanagi–Harada syndrome

C. Influence of Iridocyclitis on Intraocular Pressure

Normal IOP	Low IOP	High IOP
Iridocyclitis can be associated with normal intraocular pressure	More often, iridocyclitis causes low IOP due to ciliary shock, which recovers with the initiation of treatment	High IOP is seen in the following conditions • Trabeculitis • Annular synechiae causing the pupillary block and angle-closure • Peripheral anterior synechiae • Steroid-induced

D. Posterior Segment Signs of Iridocyclitis

1. Cystoid macular edema
2. Epiretinal membrane

2. A 44-year-old man with HIV presents with floaters and diminution of vision in his right eye. His CD4+ count is 120/mm^3.

A. Enumerate three causes for floaters. (3 marks)

B. Name three organisms that can cause posterior uveitis in a patient with HIV. (3 marks)

C. Explain the importance of knowing the CD4+ count. (3 marks)

D. Name the typical description for active ocular toxoplasmosis. (1 mark)

A. Causes of Floaters

1. Muscae voltantes (physiological)
2. Vitreous hemorrhage
3. Intermediate uveitis

B. Organisms Causing Posterior Uveitis in HIV Infected Patients

1. *Mycobacterium tuberculosis*
2. *Toxoplasma gondii*
3. Cytomegalovirus

C. Importance of knowing the CD4+ count

- The ocular manifestations are due to the infection *per se* or opportunistic infections or due to the drugs used against HIV/AIDS
- The opportunistic infections depend on the CD4+ cell count. Knowing the CD4+ count helps in expecting the organism and diagnosis

Manifestations	CD4+ cell count 0–199	CD4+ cell count 200-499	CD4+ cell count >500
Adnexal lesions	—	Blepharitis	—
Anterior segment	Ocular surface squamous neoplasia	Dry eye Kaposi sarcoma Molluscum contagiosum Herpes zoster ophthalmicus	Allergic conjunctivitis
Posterior segment	CMV retinitis Progressive outer retinal necrosis Candidal retinochoroiditis Endophthalmitis	Toxoplasmosis Intermediate uveitis Retinal vasculitis Tuberculous uveitis	HIV retinopathy Optic neuropathy

D. Typical description for active ocular toxoplasmosis

'Headlight in the fog appearance'.

3. A 24-year-old man had trauma to his right eye with a stone while using an electric motor. After two days he developed severe pain,loss of vision, and redness in the same eye

A. State the most probable diagnosis. (1 mark)

B. Explain the etiology and clinical features of this condition. (4 marks)

C. Explain the investigations and treatment of this condition. (5 marks)

A. Most Probable Diagnosis

Post-traumatic endophthalmitis.

B. Etiology and Clinical Features

Etiology

- Infective endophthalmitis
  - Bacteria (most commonly in post-operative is Staphylococcus, Strepcocci)
  - Fungi: More in vegetative injury
- Sterile endophthalmitis
  - Post-operative sterile endophthalmitis (TASS)
  - Pure copper foreign body
  - Tumor necrosis

Clinical Features

Symptoms

Pain, loss of vision.

Signs

- Lid edema
- Hazy cornea, anterior chamber reaction, hypopyon
- Edges of the wound may be swollen and gaping
- Yellow reflex in the pupil
- Exudation in the vitreous—fundus may not be visible

C. Investigations and Treatment

Investigations

- Aqueous and vitreous tap for culture and sensitivity with Gram's stain will help understand the organism
- Diagnosis is confirmed by B-scan. It shows multiple moderately echogenic opacities within the vitreous. Any associated retinal detachment may be seen

Treatment

Intravitreal Injection

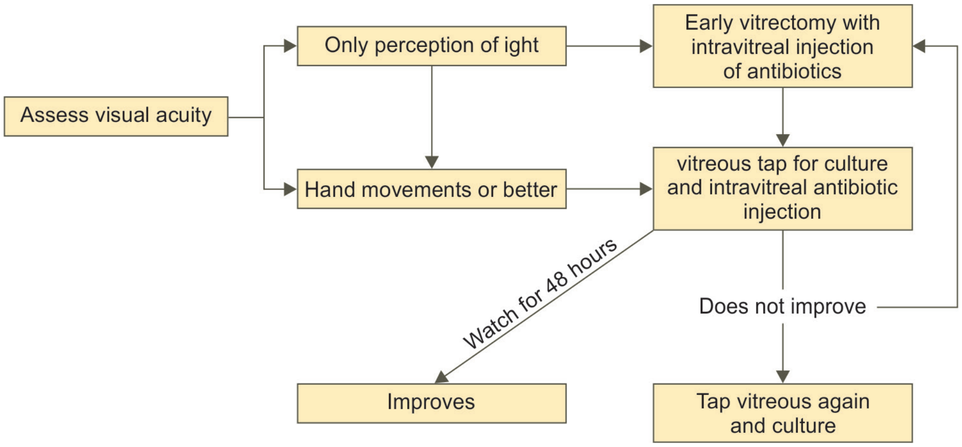
- Broad-spectrum antibiotics is given in the pars plana region (3 mm behind limbus in pseudophakic eyes, 3.5 mm behind limbus in phakic eyes)
- The usual antibiotics preferred are vancomycin (0.1 mg/0.1 ml) and ceftazidime (2.25 mg/0.1 ml)
- Other options are vancomycin + amikacin (0.4 mg/0.1 ml) and vancomycin + gentamicin (0.2 mg/0.1 ml)

Subconjunctival Injection

- Combination of antibiotics with steroids is a form of adjunct therapy
- It is given for a week to maintain intraocular concentration
    - Vancomycin 25 mg/0.5 ml + ceftazidime 100 mg/0.5 ml
    - Vancomycin + cefuroxime 125 mg/0.5 ml
- Subconjunctival dexamethasone (4 mg/ml) is given to decrease the inflammation under the cover of antibiotics

Topical Antibiotics

- Vancomycin 50 mg/ml or cefazoline (50 mg/ml) + amikacin (20 mg/ml) or tobramycin (15 mg%)
- Topical steroids like prednisolone and dexamethasone are frequently instilled
- Supportive therapy in the form of cycloplegics and antiglaucoma medications has to be started
- A study-endophthalmitis vitrectomy study (EVS) outlines the treatment protocol for postoperative endophthalmitis


Assess visual acuity
Only perception of ight
Hand movements or better
Early vitrectomy with intravitreal injection of antibiotics
vitreous tap for culture and intravitreal antibiotic injection
Watch for 48 hours
Does not improve
Improves
Tap vitreous again and culture

SHORT ESSAYS

1. Give the SUN classification of uveitis. (5 marks)

SUN Classification of Uveitis

1. **Anterior uveitis**
    i. Iritis (iris inflammation)
    ii. Anterior cyclitis (ciliary body inflammation)
    iii. Iridocyclitis
2. **Intermediate uveitis**
    i. Pars planitis
    ii. Posterior cyclitis
    iii. Hyalitis
3. **Posterior uveitis**
    i. Choroiditis (focal or diffuse)
    ii. Retinitis

  iii. Retinochoroiditis
  iv. Chorioretinitis
  v. Retinal vasculitis
  vi. Neuroretinitis

4. **Pan uveitis**
  i. Inflammation in the anterior chamber, vitreous, and retina/choroid

2. Explain the formation of keratic precipitates (KPs). Explain the classification based on appearance. (2+3 marks)

Formation of KPs

- In iridocyclitis, the corneal endothelium becomes sticky
- There are inflammatory cells in the aqueous that keep floating with the convection currents of the aqueous. They stick to the lower part of the cornea and are termed 'keratic precipitates'

Classification of KPs

Fine KPs (small, non-pigmented)	They are associated with acute nongranulomatous inflammation
Large, mutton fat, greasy KPs	They are associated with granulomatous inflammation
Pigmented, crenated KPs	They are a sign of chronic or healed anterior uveitis
Diffuse, stellate KPs	Occurrence of KPs above the horizontal midline Viral uveitis, Fuch's heterochromic iridocyclitis

3. Classify uveitis based on etiology. (5 marks)

Etiological Classifcation of Uveitis

1. **Infective uveitis**
  - It can be due to exogenous (entry of organism from the environment), secondary (spread from other ocular structures), or endogenous (through blood from distant sources)
    i. *Bacteria:* Mycobacteria, *Brucella*, *Treponemes*, streptococci, staphylococci
    ii. *Viruses:* Herpes simplex and zoster, cytomegalovirus, human immunodeficiency virus
    iii. *Parasites: Toxoplasma*, *Toxocara*, onchocerciasis
    iv. *Fungi: Candida*, *Aspergillus*, cryptococci
2. **Allergic uveitis**
  i. Microbial allergy
  ii. HLA associated: B27: ankylosing spondylosis, Reiter's; B5: Behcet's, DR4 and DW15: VKH syndrome
  iii. Autoimmune: Associated with Still's disease, rheumatoid arthritis, Wegener's granulomatosis
3. **Toxic uveitis**
  i. Endotoxins: Uveitis associated with acute pneumococcal or gonococcal conjunctivitis, keratomycosis
  ii. Endocular toxins: Uveitis associated with long-standing retinal detachment, tumors (masquerade syndromes), and phacotoxic uveitis
  iii. Exogenous toxins: Drugs, chemical irritants

4. **Traumatic uveitis**
    - It is due to:
        i. Mechanical injury
        ii. Entry of foreign body or organism
        iii. Blood
        iv. Sympathetic ophthalmia in the other eye
5. **Uveitis associated with noninfective systemic diseases**
    - Polyarteritis nodosa, SLE, diabetes, gout, etc.
6. **Idiopathic uveitis**
    - Pars planitis, Fuch's heterochromic iridocyclitis, other nonspecific idiopathic uveitis

4. Describe the anterior segment signs of iridocyclitis. (5 marks)

Cornea

- Circum corneal congestion or ciliary congestion is congestion of the anterior ciliary vessels
- Ciliary tenderness is elicited by gentle pressure over the ciliary region
- Keratic precipitates (Fine KP, mutton fat KPs)
- Band shaped keratopathy (late stages)

Iris

- Muddy iris
- Koeppe nodules: Nodules at pupillary border
- Busacca nodules: Nodules on the iris surface
- Berlin nodules: Nodule in angle
- Festooned pupil: The pupil is irregular due to patchy posterior synechiae at various positions. It is discovered on pupillary dilatation
- Ectropion pupillae: eversion of the iris surface leading to the visibility of the posterior surface of the iris. It happens due to the contraction of fibrinous exudate on the anterior surface of the iris
- Seclusio pupillae formation of posterior synechiae in an annular fashion that blocks the outflow of aqueous from the posterior chamber to anterior chamber
- Occlusion pupillae: Blocking of the pupil by exudates

Anterior Chamber

- Cells
    - A sign of activity, are seen as particles in the aqueous
    - It is graded as follows:

Grade	Cells in field
0	<1
0.5+	1–5
1+	6–15
2+	16–25
3+	26–50
4+	>50

- Flare
    - A measure of turbidity of the aqueous
    - Its grading is shown below

Grade	Description
0	None
1+	Faint
2+	Moderate (iris and lens details clear)
3+	Marked (iris and lens details lazy)
4+	Intense (fibrin or plastic aqueos)

- Plasmoid aqueous
    - Turbid aqueous when the protein concentration of aqueous is as high as plasma
- Hypopyon
    - It is formed due to collection of exudates in the anterior chamber
- Depth of the anterior chamber varies with the clinical situation

Seclusio pupillae	Shallow at the periphery, deep at center
Occlusio pupillae	Diffusely shallow
Peripheral anterior synechiae	Irregularly shallow

- Lens
    - Pigments on the anterior lens capsule
    - Posterior synechiae
    - An exudative membrane on the lens capsule
    - Complicated cataract

5. Compare and contrast granulomatous and nongranulomatous uveitis with examples. (5 marks)

Or

Explain the pathological classification of uveitis. (5 marks)

Feature	Nongranulomatous uveitis (plastic)	Granulomatous uveitis
Cause	Due to toxins, antigen, and antibody reactions. (Types II and III)	Usually due to invasion of an organism or due to type IV hypersensitivity
Pathology	Polymorphonuclear cells, lymphocytes	Giant cells, epithelioid cells, macrophages
Onset	Acute	The usually slow, indolent course
Clinical course	Short	Chronic, remissions exacerbations
Grade of inflammation	High	Low
Keratic precipitates	Many, small	Less, large mutton fat KPs
Aqueous cells and flare	Marked, numerous cells, fibrinous exudates commonly seen	Less, a few cells
Iris nodules	Absent	Present

Contd.

Feature	Nongranulomatous uveitis (plastic)	Granulomatous uveitis
Posterior synechiae	Thin and tenuous	Thick and broad-based
Posterior segment involvement	Not commonly involved	Commonly involved
Examples	Viral, HLA B27 associated uveitis, Behcet syndrome, JRA	Tuberculosis, VKH, sympathetic ophthalmia, sarcoidosis Tuberculosis can cause both granulomatous and nongranulomatous types of uveitis

6. Explain the clinical features and management of intermediate uveitis. (5 marks)

Intermediate Uveitis

- An insidious, chronic, relapsing inflammation primarily of the vitreous
- They tend to be bilateral and more in females
- Its subset, pars planitis is the term used for idiopathic inflammation
- Intermediate uveitis can be associated with multiple sclerosis (HLA DR15), Lyme's disease, and sarcoidosis. An idiopathic variant is more commonly seen in children and tends to be aggressive
- IU associated with systemic disorders occurs in the third and fourth decades

Clinical Features

Symptoms

- The most common presenting complaint is unilateral or bilateral floaters associated with diminished vision
- Metamorphopsia

Signs

- On examination, the eye may show signs of anterior uveitis, especially in those with systemic associations
- Vitreous cells and haze, more so in the anterior part, are pathognomonic for IU
- Vitreous may also be studded with exudates, called 'snowballs' appearance
- The peripheral retinal examination is mandatory in these cases, which reveal snow banking, i.e. whitish-gray exudative membrane is seen circumferentially; more so inferiorly
- Neovascularization may develop on the snowbanks
- Perivascular sheathing and periphlebitis can also be seen
- Cystoid macular edema can also be present in many cases as a result of inflammation

Management

Investigations

- FFA helps to detect peripheral neovascularization
- OCT helps to detect cystoid macular edema
- Following a thorough ophthalmic examination, systemic associations of IU as mentioned before have to rule out

Treatment

- 80% of cases resolve spontaneously, hence may not require treatment
- In severe non-resolving conditions, steroids are given in the form of subTenon injections or oral administration
- If associated with multiple sclerosis, interferon-beta may be of help
- In presence of neovascularization in the peripheral retina, laser photocoagulation is indicated
- Vitrectomy: It is indicated when there is no response to steroids, vitreous opacities significantly decreasing vision, unresolving cystoid macular edema, vitreous hemorrhage, tractional retinal detachment, and epiretinal membrane
- Accompanying endo-LASER can be done if associated neovascularization is present

7. Explain the clinical features of posterior uveitis. (5 marks)

Posterior Uveitis

- Inflammation of the choroid and/or retina
- It can be infectious or noninfectious

Clinical Features

Symptoms

- The patient usually presents with visual symptoms predominantly associated with floaters
- There is no pain or redness of the eye as such
- In cases of peripheral lesions, no symptoms may be reported and hence tend to be accidentally found on routine examination
- The other symptoms are metamorphopsia (distorted images), macropsia (due to increase in distance between photoreceptors), micropsia (due to crowding of photoreceptors), photopsia (sensation of flashes of light), and positive scotoma

Signs

- One may find a few KPs and the anterior segment is usually quiet
- Fundus examination may reveal vitreous cells and flare
- Vitritis is more commonly seen with retinitis lesions
- Fundus examination shows retinitis/choroiditis/retinal vasculitis/a combination of these in the form of focal, multifocal, or diffuse lesions.
  - The retinitis patch looks yellowish bright spot with fuzzy margins, obscuring the blood vessels
  - Choroiditis is seen as pale yellow subretinal lesion with normal overlying blood vessels
  - Retinal vasculitis is seen sheathing of veins or arteries or both. There may be associated hemorrhages or capillary occlusion
- After resolution, either due to therapy or remission
  - Retinitis lesion becomes atrophic, thinned out and translucent
  - Choroiditis lesion heal by pigmentation. The vessels which run through that segment appear thin and sclerotic. Sclera may be visible below and may simulate a coloboma
- In cases of severe posterior uveitis, complications like complicated cataracts, choroidal neovascularization, secondary glaucoma, and rhegmatogenous retinal detachment can occur

- Some of the important patterns of posterior uveitis are listed below:

Congenital toxoplasmosis	Usually bilateral punched out lesions simulating macular coloboma
Acquired toxoplasmosis	Headlight in the fog appearance: intense vitreous haze with creamy yellow retinitis patch Arising from the edge an old healed lesion
CMV retinitis	A brush-fire pattern of full-thickness retinal necrosis with hemorrhages and exudation
Tuberculosis	Choroid tubercles (yellowish subretinal lesions of varying size) usually associated with meningitis, tubercular conglomerate granuloma (solitary) Serpiginous-like choroiditis: centrifugal spread of choroiditis with healing seen in the other end
Sarcoidosis	Choroidal and retinal granulomas, periphlebitis retinae: sheathing around veins resembling ‘candle wax drippings
Toxocariasis	Chronic endophthalmitis: Presents with leukocoria due to marked vitreous clouding, in children of the age of 2–10 years, poor prognosis Posterior pole granuloma: A yellow-white, round, solitary, raised nodule, mild inflammatory response, about 1–2 disc diameter in size, located either at the macula or in the centrocaecal area, in children of age 5–15 years, presents with decreased vision Peripheral granuloma: Can have vitreous bands and develop retinal detachment

8. Describe the etiology, clinical features, and management of sympathetic ophthalmia. (1+2+2 marks)

Or

A 56-year-old woman presents with left-sided ocular pain and redness. Granulomatous panuveitis is diagnosed in the left eye. She gives a history of penetrating injury and loss of vision in the right eye. State the diagnosis. Explain the signs and management of this condition. (1+2+2 marks)

Sympathetic Ophthalmia

A bilateral serious, chronic granulomatous, panuveitis due to sensitization to choroidal antigen due to either injury or surgery in the fellow eye.

Predisposing Factors and Etiology

1. Injury in the ciliary region
2. Penetrating injury/vitreoretinal surgery
3. Incarcerated ciliary body/lens capsule or iris
4. Can occur in any age group
    - It is thought to be due to a T cell-mediated response driven against sensitized uveal antigens (Fig. 6.1.1)
    - Viral infection may be initiating factor
    - The eye which is traumatized is called the exciting eye
    - The other eye is called the sensitizing eye

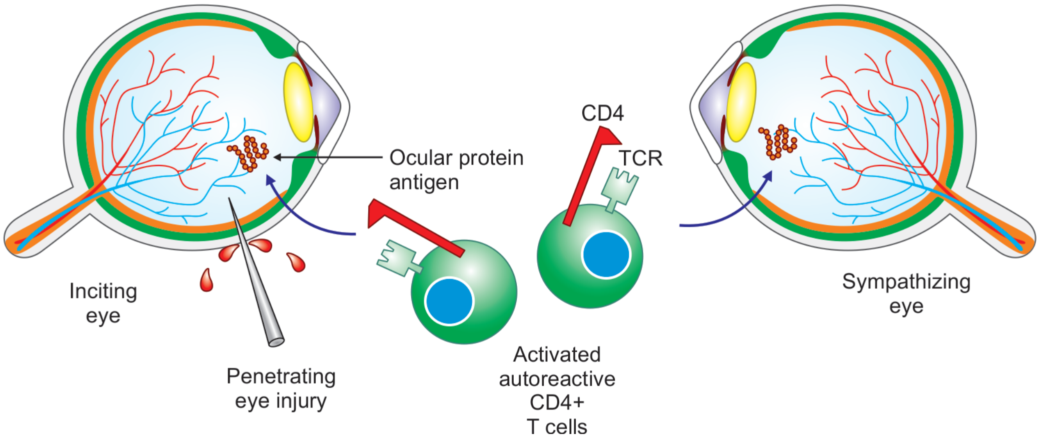


Fig. 6.1.1: Pathogenesis of sympathetic ophthalmia

Clinical Features

Prodromal Stage

- When the sympathetic ophthalmia begins, there is a return of ciliary congestion, tenderness, and lacrimation in the shrunken eye
- In the sympathizing eye, the first symptom to begin is photophobia and defective near vision as a result of the weakness of accommodation
- A careful examination may reveal ciliary tenderness, KPs, vitreous cells, and flare
- Fundus examination reveals Dalen–Fuch nodules (yellow subretinal lesions)

Established Stage

- Full-fledged granulomatous uveitis features may be seen: Iridocyclitis, peripheral retinal exudation, Dalen–Fuch nodules, and papillitis
- It tends to be chronic and may last many months
- Inflammation may be severe enough to cause complications like phthisis

Prophylaxis

- Meticulous repair of any penetrating injury to take care of the uveal tissue not to be incarcerated in the wound
- Uncontrolled uveitis in the severely damaged eye for more than 2 weeks should be enucleated

Treatment

- Early recognition of signs of inflammation and starting steroid therapy in the form of intravenous methylprednisolone 1000 mg followed by oral prednisolone 1 mg/kg and topical steroids and cycloplegics
- After the eye quietens down, a maintenance dose of 15–20 mg is administered for many months and the eye has to be monitored
- Enucleation of the exciting blind eye is proposed by some but must be done before the initiation of inflammation

9. Explain various complications of uveitis. (5 marks)

Complications of uveitis can be due to inflammation per se and due to the treatment of uveitis.

Complications due to Inflammation

1. **Cornea**
    - Band keratopathy: It usually occurs in chronic inflammation
2. **Angle**
    - Peripheral anterior synechiae form between the peripheral iris and the cornea that bridge the angle and cause a block of aqueous outflow
3. **IOP**
    - Hypotony and hypotonous maculopathy occur as a result of ciliary shock and ciliary shutdown
    - Raised intraocular pressure can occur as a result of trabeculitis and scarring of the trabecular meshwork
4. **Iris**
    - Posterior synechiae form between the lens and posterior surface of the iris
    - Iris bombe forms due to annular synechia that causes forward displacement of the peripheral iris causing secondary angle-closure glaucoma
5. **Lens**
    - Complicated cataract, usually seen as posterior subcapsular cataract with bread-crumb appearance
    - Poor dilatation during cataract surgery occurs due to thinning of the iris and formation of posterior synechiae
    - Cyclitic or retrolental membrane forms due to the organization of exudates behind the lens
6. **Retina**
    - Cystoid macular edema is the most common retinal complication associated with uveitis. It is a frequent cause of diminished vision
    - Epiretinal membrane
    - Retinal detachment (exudative)
    - Subretinal fibrosis
7. **Phthisis bulbi**
    - Occurs as a result of chronic inflammation leading to ciliary body shut down leading to low IOP and complete loss of vision

Complications due to Treatment

1. **Steroids**
    - Steroid-induced glaucoma
    - Steroid-induced cataract

10. Describe the etiology, clinical features, and management of Pan ophthalmitis. (5 marks)

Pan ophthalmitis

Inflammation of all the coats of the eyeball along with orbital tissues.

Etiology

- Exogenous entry of organisms can follow penetrating injury or postoperatively. The usual organisms implicated are gram-positive cocci
- Endogenous infection is seen in severely immunocompromised individuals with indwelling catheters and prostheses

Clinical Features

Symptoms

- Severe eye pain
- Complete loss of vision

Signs

- On examination, visual acuity is negative to the perception of light
- The lids are edematous, the eyeball is proptosed and extraocular movements are restricted due to orbital edema
- Conjunctival chemosis and congestion are present
- The cornea is usually sloughing off with an anterior chamber full of purulent material
- Fundus cannot be visualized. Intraocular pressure is raised

Management

- Evisceration is the treatment of choice. Supportive systemic antibiotics and anti-inflammatory drugs are given
- Evisceration is the removal of the entire contents of the eyeball leaving behind the sclera
- Evisceration can be done in two methods: Simple and frill evisceration
- In the former, the whole sclera is left behind

SHORT ANSWERS

1. Explain the clinical features of plastic iridocyclitis. (3 marks)

Plastic Iridocyclitis

- Another name for nongranulomatous iridocyclitis
- It is usually acute and is associated with intense inflammation

Clinical Features

Symptoms

- Patients present with acute onset of redness, periocular pain, and photophobia
- Visual acuity depends on the severity of the inflammation

Signs

- The eye is congested, with dilated ciliary vessels. Ciliary tenderness is usually present
- The cornea shows fine, nonpigmented KPs in the inferior aspect of the cornea. Corneal edema may be present
- In the anterior chamber, there is a reaction in the form of cells and flare. In non-granulomatous uveitis, the reaction tends to be intense with 'plasmoid aqueous'. The protein content of the aqueous may be as that of plasma
- The iris is muddy and engorged. The pupil is usually miotic
- Hypopyon is usually present

2. Explain the terms (3 marks)

A. Occlusio pupillae
B. Seclusio pupillae
C. Iris Bombe

A. Occlusio Pupillae

Occlusion of the pupillary aperture due to exudates.

B. Seclusion Pupillae

Occlusion of the aqueous outflow through the pupil due to formation of annular or ring synechiae circumferentially.

C. Iris Bombe

- When annular synechiae forms, the aqueous cannot escape through the pupil.
- As a result, it builds up in the posterior chamber and pushes the peripheral iris towards the angle.
- This causes bulging of the peripheral iris forward to cause 'iris bombe' (Fig. 6.1.2)

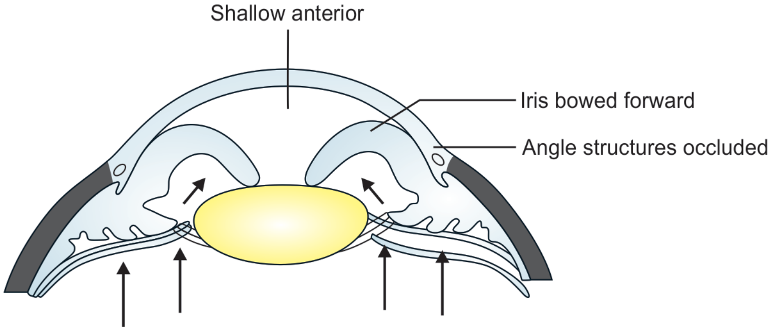


Fig. 6.1.2: Iris bombe

3. Explain the pupillary changes that can occur in uveitis. (3 marks)

- The pupil becomes small and the reaction becomes sluggish in acute iritis
- Iris nodules
  - It may be studded with an inflammatory nodule at the border (Koeppe's nodules) or the collarette (Busacca's nodules) as seen in granulomatous inflammation.
  - The Koeppe's nodules form posterior synechiae
- The pupil may be irregular due to patchy posterior synechiae at various positions (Festooned pupil, Fig. 6.1.3)
  - It is discovered after instilling mydriatics.
  - The adhered part of the iris does not dilate whereas the free iris moves behind
- Ectropion pupillae
  - Eversion of the iris surface leading to the visibility of the posterior surface of the iris
  - It happens due to the contraction of fibrinous exudate on the anterior surface of the iris

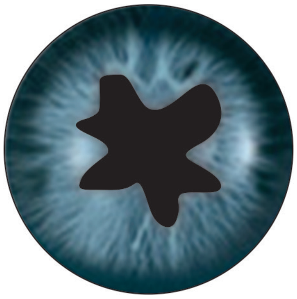


Fig. 6.1.3: Festooned pupil

4. Justify the reason for keratic precipitates being present in the inferior half of the cornea. Name one condition where this rule is not followed. (2+1 marks)

KPs are seen on the inferior half of the cornea

- Keratic precipitates are a sign of uveitis. In uveitis, the endothelium of the cornea becomes sticky
- As a result, polymorphonuclear cells that are released into the aqueous get attached to the endothelium
- The anterior of the eye is relatively cooler than the posterior due to exposure to air and evaporation of the tear film. As a result convection currents are set up in the aqueous. The aqueous that is closer to the cornea, sinks and the aqueous that is near the iris rises (Fig. 6.1.4)
- During this process, the keratic precipitates settle down in the form of an 'Artl's triangle'

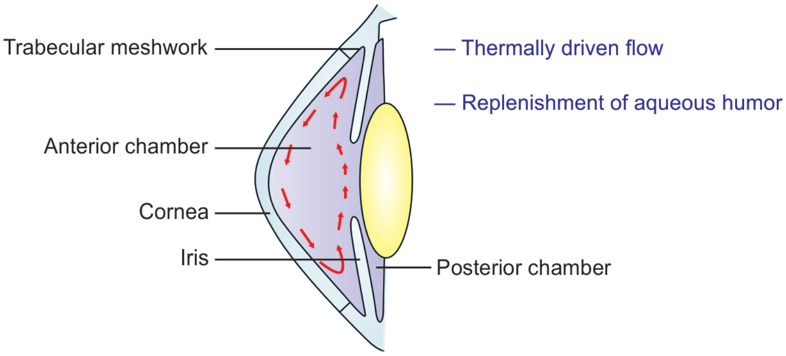


Fig. 6.1.4: Convection currents

Condition in which this Rule is not Followed

Stellate diffuse KPs are characteristically seen in herpetic iridocyclitis and Fuch's heterochromic cyclitis

5. Compare and contrast atrophic bulbi and phthisis bulbi. (3 marks)

- Atrophic bulbi is the initial stage of phthisis bulbi
- Phthisis bulbi is also known as atrophic bulbi with disorganization.

Similarities	Differences
• Both have no visual acuity • The lens is cataractous • The retina is atrophic and detached • The eye is smaller than usual and IOP is low	• Phthisis bulbi is a shapeless eye and does not have a structure, whereas atrophic bubli still maintains the original eyeball shape • Phthisis bulbi is associated with calcifications in all parts of the eye evident on CT scan or B scan. There is bone metaplasia evident in RPE • The cornea becomes sclera-like and the sclera becomes markedly thickened in phthisis bulbi. The parts of the eyeball cannot be differentiated from one another

6. Enumerate three indications of evisceration. (3 marks)

1. Expulsive choroidal hemorrhage
2. Panophthalmitis
3. Bleeding anterior staphyloma

COMPETENCY

OP6.2 IDENTIFY AND DISTINGUISH ACUTE IRIDOCYCLITIS FROM CHRONIC IRIDOCYCLITIS

SHORT ESSAY

1. Differentiate between acute and chronic iridocyclitis. (5 marks)

Differentiating Features

Parameter	Acute Iridocyclitis	Chronic Iridocyclitis
Symptoms	• Acute onset of pain, brow ache, and hazy vision • Lasts for about 6 weeks to 3 months	• Chronic indolent course, with acute exacerbations • Diminished vision is a more common presentation • Duration >3 months
Signs	• Ciliary congestion and tenderness • Corneal edema • Fresh KPs • Anterior chamber cells and flare, • Exudative membrane, hypopyon may or may not be there	• No congestion • Old crenated KPs • Anterior chamber flare is more in comparison with cells • Presence of posterior synechiae • A complicated cataract may be present • Peripheral anterior synechiae and glaucoma may be present
Complications	• Hypertensive uveitis • Ciliary shock and hypotony • Disc edema and hypotonus maculopathy • Choroidal detachment	• Complicated cataract • Annular synechiae and pupillary block glaucoma • Retinal detachment due to contraction of fibrovascular membrane • Neovascular glaucoma • Phthisis bulbi • Band-shaped keratopathy
Pathology	• Usually nongranulomatous response	• May be granulomatous or nongranulomatous
Examples	• Idiopathic anterior uveitis • Traumatic uveitis • Bacterial endophthalmitis • HLA B27 associated iridocyclitis	• Cyclitis • Intermediate uveitis • Sympathetic ophthalmia • VKH syndrome • Tuberculous uveitis • Syphilitic uveitis • Stills disease or juvenile idiopathic arthritis-associated uveitis

SHORT ANSWERS

1. Define (1 mark each)

A. Acute uveitis
B. Chronic uveitis
C. Recurrence of uveitis

A. Acute Uveitis

Onset is sudden and duration of symptoms and signs of uveitis last not more than 3 months.

B. Chronic Uveitis

Insidious onset, duration of disease lasting for more than 3 months.

C. Recurrent Uveitis

Repeated attacks of uveitis separated by periods of inactivity of more than 3 months without treatment.

2. Enumerate three causes for chronic uveitis. (3 marks)

1. Fuch's heterochromic cyclitis
2. JIA associated uveitis
3. Syphilitic uveitis

3. Describe three signs of chronic uveitis. (3 marks)

Signs of Chronic Uveitis

1. **Visual acuity**
    - Maybe subnormal owing to sequelae of uveitis
2. **Cornea**
    - Old crenated KPs
    - Band keratopathy
3. **Anterior chamber**
    - Flare is more common than cells and is out of proportion to cells
    - Depth may be uneven due to the formation of anterior synechiae
4. **Iris**
    - Areas of iris atrophy may be present
5. **Lens**
    - Complicated cataract
    - Pigments on the lens
6. **Retina**
    - Subretinal fibrosis
    - An epiretinal membrane may be present
    - Chronic macular edema

COMPETENCY

OP6.3 ENUMERATE SYSTEMIC CONDITIONS THAT CAN PRESENT AS IRIDOCYCLITIS AND DESCRIBE THEIR OCULAR MANIFESTATIONS

SHORT ESSAYS

1. A 36-year-old man with back pain and stiffness presented with recurrent episodes of iridocyclitis. On further investigating, an X-ray of the lumbar spine shows a "bamboo-spine" appearance.

A. Which systemic condition is being described here and what is the HLA type expected? (2 marks)

B. Enumerate other two systemic manifestations of the same HLA type with ocular manifestations. (1 marks)

C. Annotate the management of this condition. 2 marks)

A. The Systemic Condition Described Here is

- Ankylosing spondylosis
- It is associated with HLA B27

B. Other Conditions Associated with HLA B27

1. Reiter's disease
2. Psoriatic arthritis

C. Management

- Uveitis associated with HLA B27 is usually unilateral, acute, and anterior
- It is treated with topical steroids and cycloplegics
- The cycloplegics have to be continued for three weeks after control of inflammation to decrease recurrence
- In case of recurrent disease, systemic NSAIDs and monoclonal antibodies should be used

2. Enumerate four systemic conditions that are associated with anterior uveitis. Describe the features of Heerfordt syndrome. (4+1 marks)

Systemic Conditions That are Associated with Anterior Uveitis

1. Ankylosing spondylosis
2. Reiter's disease
3. Inflammatory bowel disease
4. Tubulointerstitial nephritis

Heerfordt's Syndrome

- It is also known as uveoparotid fever and is characterized by bilateral granulomatous panuveitis + painful enlargement of parotid glands + cranial nerve palsies + skin rashes + fever
- It is seen as an acute manifestation of Sarcoidosis

3. Name the systemic autoimmune disorder in which the eye develops a recurrent "cold hypopyon"? Describe other ocular manifestations of that disease. (1+4 marks)

Systemic Autoimmune Disorder is

- Cold hypopyon is seen characteristically in Behcet's syndrome
- It is characterized by recurrent episodes of anterior uveitis with hypopyon with minimal signs of inflammation like cells and flare. So, it is described as a 'cold hypopyon'

Ocular manifestations of Behcet's Syndrome

- Behcet's disease is an idiopathic multisystem disorder characterized by obliterative arteritis affecting vessels of all sizes
- The uveitis is typically bilateral and anterior. Iridocyclitis is recurrent and associated with hypopyon
- There is minimal inflammation in the form of cells and flare
- Posterior uveitis is characterized by vitritis, periphlebitis retinae, and retinitis that can present as white creamy exudates. It can also cause panuveitis

4. Describe the manifestations of tuberculosis in the eye. (5 marks)

- Ocular manifestations of tuberculosis are due to hematogenous spread of the organism or due to a hypersensitivity triggered by tubercular protein.
- It has varied manifestations from the ocular surface to the retina

Lid	• Lupus pernio, cold abscess (rare)
Conjunctiva	• Granuloma can occur in the conjunctiva • Phlyctenular conjunctivitis is a type IV hypersensitivity reaction to tubercular protein and is commonly seen in children. As a result, there is a formation of a pinkish nodule with feeding blood vessels. They respond well to topical steroids
Sclera	• Scleritis can occur as a hypersensitivity reaction • A scleral ulcer is an infectious process
Cornea	• Interstitial keratitis is a result of a hypersensitivity reaction
Uvea	• Anterior uveitis: It is usually granulomatous with mutton-fat KPs and iris nodules • Intermediate uveitis is characterized by vitritis and snow banking • Choroid tubercles, granuloma, serpiginous-like choroiditis are posterior segment manifestations of TB • Choroid tubercles are subretinal lesions of smaller dimensions usually seen in the posterior pole. They are seen in association with military tuberculosis and CNS tuberculosis
Retina	• Infectious retinitis • Eales' disease is a hypersensitivity reaction characterized by recurrent vitreous hemorrhages and perivenous exudates (retinal vasculitis)
Optic nerve	• Papilledema due to arachnoiditis and noncommunicating hydrocephalus

5. Annotate the ocular manifestations caused by this organism. (5 marks)

Or

Enumerate the manifestations of syphilis in the eye. (5 marks)

- Syphilis has many forms of ocular presentation and thus is known as the great masquerader”

Stages of Syphilis and Ocular Manifestations

Primary	• Chancres in eyelid and conjunctiva (rare)
Secondary	• Interstitial keratitis (with ghost vessels – salmon patch) • Scleritis • Cataract • Acute non granulomatous iritis (may be as a part of Jarisch–Herxheimer reaction) • Chronic granulomatous uveitis • Acute syphilitic placoid posterior chorioretinitis (ASPCC) • Optic neuritis, papilledema
Latent	• None
Tertiary	• Iris roseola (dilated blood vessels in the iris due to microembolism of vessels due to organisms) • Gumma of the uveal tract • Argyll Robertson pupil
Congenital syphilis	• Hutchison's triad: Interstitial keratitis + Hutchison's teeth + sensorineural deafness • Developmental cataract

6. Describe the clinical features of congenital and acquired ocular toxoplasmosis. (5 marks)

- Ocular toxoplasmosis is caused by *Toxoplasma gondii*
- It can be congenital or acquired

Congenital Toxoplasmosis

- Congenital toxoplasmosis is acquired by the fetus when the expectant mother suffers an episode of toxoplasmosis which may not be very apparent clinically
- When the infection happens in the first trimester, the fetus usually does not survive
- Congenital toxoplasmosis is characterized by convulsions, calcifications, and choroiditis
- The choroiditis is typically at the macula owing to the pattern of the ocular blood supply in the fetus. When discovered, the lesions are usually healed, bilateral, and appear as punched-out lesions with pigmentation. The children usually present with nystagmus and decreased central vision

Acquired Toxoplasmosis

- It may be due to *de novo* acquisition of infection due to consumption of undercooked meat or reactivation of old scars where the worms remain as bradyzoites
- They are usually seen in moderately immunosuppressed conditions
- Commonly, a retinitis lesion is seen to begin from the edge of old scars (recurrent infection). The parasites replicate and cause an intense vitreous reaction
- The lesion is yellowish raised plaque and appears as a "headlight in the fog". In extreme immunocompromise, vitritis may be less

7. Enumerate four opportunistic infections that cause posterior uveitis in immunocompromised individuals. Discuss the clinical features of CMV retinitis. (2+3 marks)

Opportunistic Infections That Cause Posterior Uveitis

1. Toxoplasmosis
2. CMV retinitis
3. Posteriori uveitis caused by tuberculosis
4. Candidal endophthalmitis

CMV Retinitis

- Most frequent opportunistic infection in immunocompromised individuals
- Incidence has decreased after the initiation of HAART
- Most commonly seen when CD4+ counts <50/mm^3

Symptoms

- The patients present with floaters and diminution of vision depending on the amount of retinal involvement
- Many-a-times, they can be asymptomatic when the lesions are in the extreme periphery and not associated with significant vitritis

Signs

- The anterior segment is usually quiet
- A careful fundus examination of both eyes by mydriasis is necessary

Clinical Forms

1. **Fulminant form:** Brush-fire pattern, seen as hemorrhagic vasculitis and necrosis of retina along the major arterial arcade. This is a more fulminant variant and has more impact on visual acuity
2. **Granular form or indolent form:** Seen as granular lesions in the periphery. Sometimes, there is severe perivascular exudation leading to a 'frosted branch' angiitis-like appearance

SHORT ANSWERS

1. Describe the features of Vogt–Koyanagi–Harada syndrome. (3 marks)

- Vogt–Koyanagi–Harada syndrome is an idiopathic multisystem disorder
- Associated with HLA DR4 and DW15
- Ocular + cutaneous + CNS manifestations
- The disease occurs in many phases

Prodromal Phase

- Flu-like symptoms: fever, headache
- Tinnitus, hearing loss

Uveitic Phase

- Bilateral anterior granulomatous uveitis characterized by the formation of iris nodules and posterior synechiae
- Posterior uveitis with multifocal chorioretinitis lesions with bilateral exudative detachment
- Increased choroidal thickness

Chronic Recurrent Phase

- Chronic unteated posterior uveitis
- Depigmentation of the RPE (sunset glow fundus)
- Depigmentation of the limbus (Siguara sign)

Cutaneous Phase

- Alopecia, poliosis and vitiligo.

2. Describe the posterior segment manifestations of tuberculous uveitis. (3 marks)

- Tuberculosis can affect both the anterior and posterior segments
- The posterior segment manifestations of tuberculosis include

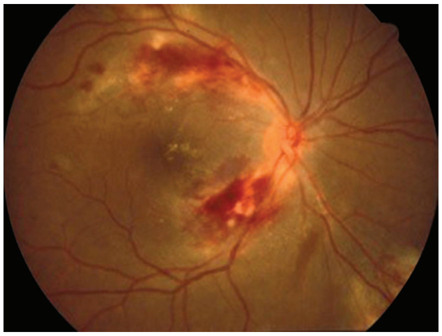
1. **Choroid tubercles**
   - These are usually multiple, subretinal, yellow flat lesions that are dispersed in the post-equatorial region and are associated with meningeal tuberculosis and disseminated tuberculosis
   - They are 1/6th to half the disc diameter size
2. **Solitary granuloma/choroidal granuloma**
   - Occurs as a focal lesion in any part of the retina. It appears yellow and well-demarcated and a size of more than 2 disc diameters
3. **Multifocal choroiditis**
   - Can also be seen in tuberculosis

4. **Serpiginous-like choroiditis**
    - There is centripetally advancing choroiditis that affects the posterior pole mainly
    - The lagging edge shows features of healing in the form of pigmentation
    - If not treated, there is diffuse chorioretinal atrophy

3. Summarize the treatment of CMV retinitis.

Or

A 32-year-old man with HIV presents with blurred vision. His CD4 count is 4/mm^3. Fundus shows the following features. (3 marks)



Summarize the Treatment of this Condition

Treatment of CMV Retinitis

- In the pre-HAART era, the only treatment was lifelong antivirals. But the quality of life has improved significantly after the introduction of HAART
- Prompt initiation of HAART is quintessential for treating CMV retinitis
- Antivirals that are recommended are ganciclovir (first line), valganciclovir, foscarnet, and cidofovir; and they are continued till there is a doubling of CD4+ counts or if CD4+ counts cross 100/mm^3 and maintained for 6 months

Drug	Induction dose	Maintenance dose
Injection ganciclovir	5 mg/kg twice daily intravenous for 3 weeks	5 mg/kg once daily IV
Oral valganciclovir	900 mg twice daily for 3 weeks	9000 mg once daily
Oral foscarnet	90 mg/kg BD for 14 days	90–120 mg/kg OD

- Other options include intravitreal injections and vitreal implants that release ganciclovir into the vitreous. They are helpful in unilateral cases
- The systemic side effects of these drugs include myelosuppression, renal dysfunction

4. Describe the management of tuberculous uveitis. (3 marks)

Management of Tuberculous Uveitis

Investigations

- In presence of features of tuberculous uveitis and a history suggestive of exposure to a patient with active tuberculosis, the following tests help to support the diagnosis of tuberculous uveitis
    - Positive tuberculin test (>10 mm induration) or positive interferon-gamma release assay
    - Chest X-ray suggestive of active or healed tuberculous lesions
    - Intraocular fluid (aqueous) PCR for tuberculous DNA
- IGRA or interferon-gamma release assay (commonly known as quantiferon) is a quantitative assay for measuring the amount of gamma interform released by the patient’s WBC in response to exposure to tubercular proteins and peptides. It cannot differentiate between latent infection and active infection
- Other possible sites of tuberculosis should be investigated by relevant imaging techniques like MRI brain, CT chest, MRI spine, abdominal and pelvic ultrasound

Treatment

Topical

- Steroids (in anterior uveitis)
- Cycloplegics
- Periocular steroids in intermediate and posterior uveitis

Systemic

- ATT for 6 months followed by isoniazid and rifampicin for another 6 months
- Systemic steroids are indicated in cases with severe inflammation

5. Enlist the clinical features of syphilitic uveitis. (3 marks)

Treponemes can affect all the coats of the eyeball.

Anterior Uveitis

- Characterized by the formation of iris roseola (dilated vessels) caused by treponemes-causing emboli of the microvasculature of the iris
- Acute nongranulomatous iritis (may be as a part of Jarisch–Herxheimer reaction)
- Chronic granulomatous uveitis is characterized by mutton fat KPs and iris nodules

Intermediate uveitis

Seen as dense vitritis.

Posterior uveitis

- Deep chorioretinitis may be focal/solitary or multifocal or diffuse. It is seen as a yellow gray subretinal lesion with surrounding shallow retinal detachment and vitrits
- Acute syphilitic placoid posterior chorioretinitis (ASPPC) with typical leopard skin pigmentation of the RPE seen during fluorescein angiography
- Necrotizing retinitis (mimics acute retinal necrosis)

Panuveitis

Inflammation of all parts of uvea

COMPETENCY

OP6.4 DESCRIBE AND DISTINGUISH HYPHEMA AND HYPOPYON

SHORT ESSAYS

1. Define hyphema. Enumerate four causes for the same. (1+4 marks)

Definition of Hyphema

Blood in the anterior chamber

Causes

1. Blunt ocular trauma
2. Hemophilia
3. UGH syndrome due to ACIOL implantation
4. Fuch's heterochromic iritis

2. Explain the clinical features of hyphema. (5 marks)

Clinical Features of Hyphema

Symptoms

- Usually hyphema follows blunt trauma

- Blurred vision
- Ocular pain
- Photophobia

Signs

- There is blood in the anterior chamber and depending on its severity, it is graded as follows
  1. Microscopic hyphema: The absence of gross collection of blood, RBCs are seen floating in the aqueous
  2. Grade 1: <1/3 of the anterior chamber filled with blood
  3. Grade 2: 1/3–1/2 of the anterior chamber filled with blood
  4. Grade 3: 1/2 to a near-total anterior chamber filled with blood
  5. Grade 4: total hyphaema or 8-ball hyphema
- IOP is usually raised
- Other features of trauma like mydriasis, subluxation of the lens, sphincter tears, and iridodialysis may be seen

SHORT ANSWERS

1. Explain the management of hyphema. (3 marks)

- The patient is advised bed rest in a propped-up position so that the cells settle down and vision clears
- Topical steroids and cycloplegics are given to decrease intraocular inflammation
- Antiglaucoma medications are given to decrease intraocular pressure as hyphema is usually associated with raised IOP. Drugs like beta-blockers, alpha agonists, carbonic anhydrase inhibitors are useful. Cholinergic and prostaglandins should not be used
- Antifibrinolytics like e-amino caproic acid and tranexamic acid can be given to prevent rebleed
- Surgical treatment is required only if IOP is very high or the hyphema does not dissolve even after 7 days. The clot is evacuated by paracentesis

2. Enumerate three causes of hypopyon. (3 marks)

1. Corneal ulcer
2. Iridocyclitis
3. Endophthalmitis

3. Define a pseudohypopyon. Differentiate it from a true hypopyon. (3 marks)

Pseudohypopyon

A noninflammatory collection of cells in the anterior chamber that resembles a hypopyon.

Differentiation from True Hypopyon

- The most common cause of a pseudohypopyon is intraocular malignancies like retinoblastoma, intraocular lymphoma, and leukemias. It may also follow an intravitreal injection of triamcinolone
- It is more convex in configuration cf hypopyon. It may not be associated with corcumcorneal congestion, anterior chamber reaction, and flare
- Also, it persists despite the use of topical steroids and does not regress

4. Differentiate between hypopyon seen in bacterial and fungal keratitis. (3 marks)

- Hypopyon is a feature of both bacterial and fungal keratitis
- Hypopyon occurs more commonly in fungal keratitis than bacterial keratitis
- However, in fungal keratitis, the hypopyon is not sterile as the fungal hyphae penetrate the cornea
- Also, its size is out of proportion to the size of the ulcer in fungal keratitis and is relatively immobile

COMPETENCY

OP6.5 DESCRIBE AND DISCUSS THE ANGLE OF THE ANTERIOR CHAMBER AND ITS CLINICAL CORRELATES

LONG ESSAYS

1. A. Define the angle of the anterior chamber. (1 mark)
B. Explain the measurement of the angle of the anterior chamber. (3 marks)
C. Describe the formation of aqueous. (3 marks)
D. Describe the outflow of aqueous. (3 marks)

A. Angle of the Anterior Chamber

- The space between the peripheral part of the cornea and the base of the iris
- It is the site of aqueous drainage

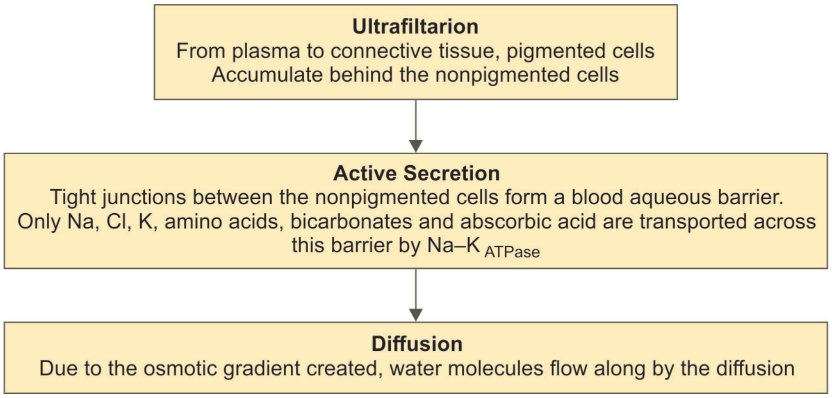
B. Measurement of the Angle of the Anterior Chamber

- The depth of the anterior chamber can be measured by gonioscopy
- It is measured based on the structures seen on gonioscopy

Grade 0	No angle structures visible	Closed	0°
Grade I	Schwalbe's line	High chance of angle-closure-very narrow-angle	10°
Grade II	Schwalbe's line, trabecular meshwork	Angle-closure possible-narrow-angle	20°
Grade III	Schwalbe's line, trabecular meshwork, scleral spur	Open-angle-no chance of angle-closure	20–35°
Grade IV	Schwalbe's line, trabecular meshwork, scleral spur, ciliary body band	Wide-open angle-no chance of angle-closure	35–45°

C. Formation of Aqueous

- It is secreted from the pars plicata of the ciliary body



Ultrafiltarion
From plasma to connective tissue, pigmented cells
Accumulate behind the nonpigmented cells

Active Secretion
Tight junctions between the nonpigmented cells form a blood aqueous barrier.
Only Na, Cl, K, amino acids, bicarbonates and abscorbic acid are transported across this barrier by Na–K $_{\text{ATPase}}$

Diffusion
Due to the osmotic gradient created, water molecules flow along by the diffusion

- The following mechanisms are involved in aqueous production. Each villus has a vascular core with a fibrous core
- Aqueous is formed by diffusion, ultrafiltration, and active secretion
- The rate of formation is 2.3 µl/min

D. Outflow of Aqueous

- The aqueous flow out into the venous system of the eye through the angle of the anterior chamber
- The angle is formed at the intersection of the cornea and the root of the iris
- The aqueous is drained by two methods (Fig. 6.5.1)

1. **Conventional pathway**
   - Trabecular meshwork → Schlemm's canal → Collector channels → Aqueous veins → Episcleral veins (90% drainage)
   - Trabecular meshwork (Fig. 6.5.2)
   - It is a sieve-like structure situated at the angle of the anterior chamber
   - It consists of three parts:
     - i. Uveal meshwork
       - ♦ Anterior-most with a diameter of 25–75 µm
       - ♦ Extends from the root of the iris to Schlemm's canal
       - ♦ Consists of large pores
     - ii. Corneoscleral meshwork
       - ♦ Extends from the scleral spur to Schwalbe's line
       - ♦ Consists of pores a little smaller (5–50 µm)
     - iii. Juxtacanalicular meshwork
       - ♦ A continuation of the corneal endothelium
       - ♦ Offers the highest amount of resistance

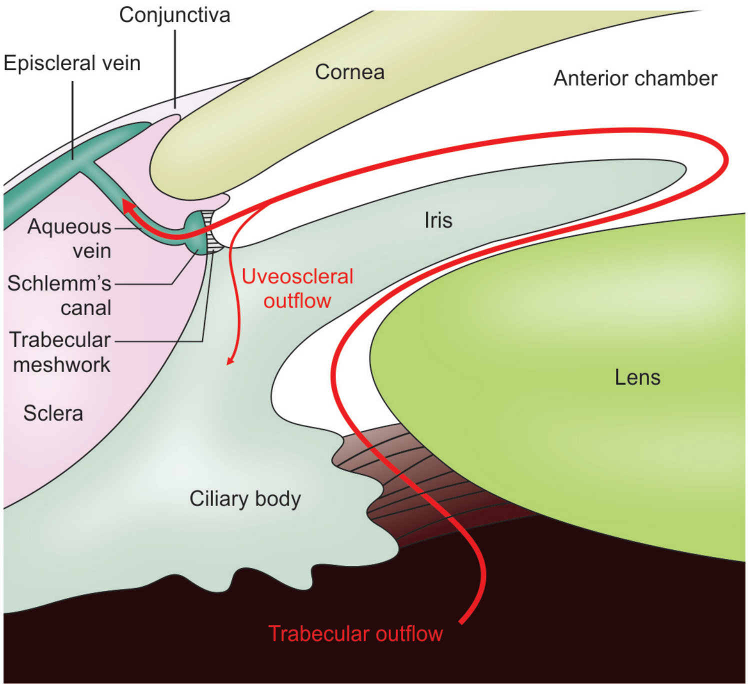

Conjunctiva
Episcleral vein
Cornea
Anterior chamber
Aqueous vein
Iris
Schlemm's canal
Uveoscleral outflow
Trabecular meshwork
Lens
Sclera
Ciliary body
Trabecular outflow

Fig. 6.5.1: Outflow of aqueous by conventional and uveoscleral pathway

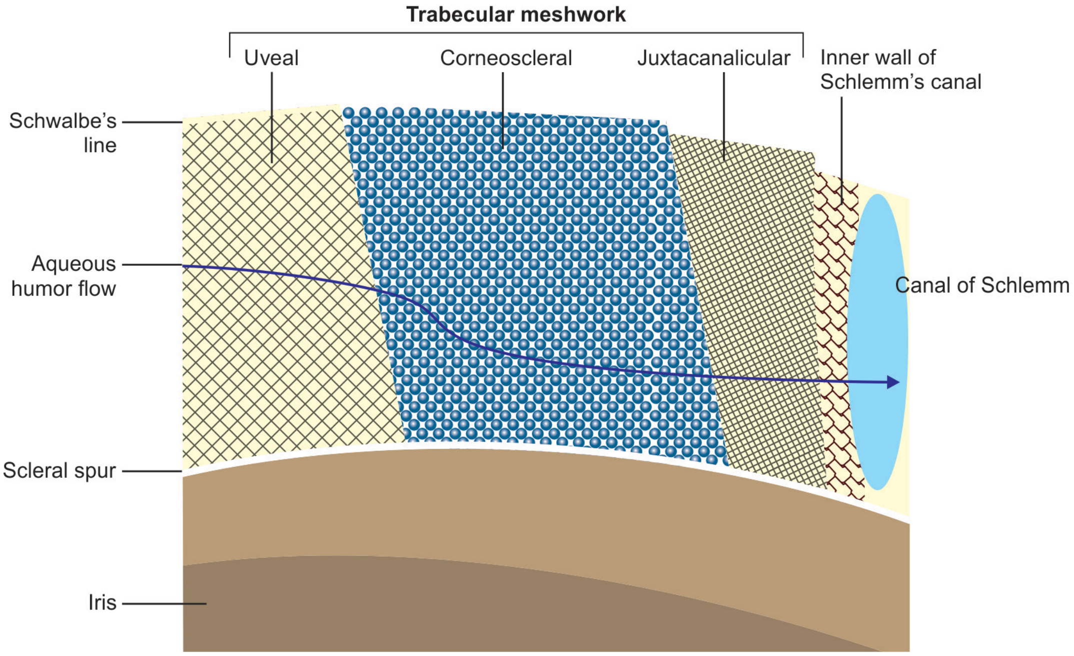
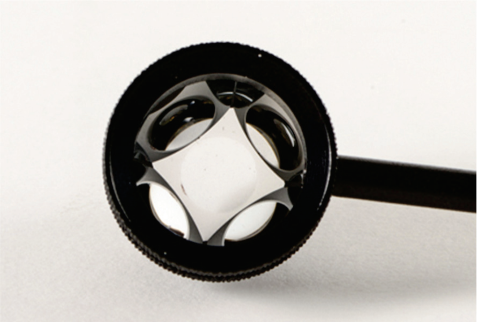

Trabecular meshwork
Uveal
Corneoscleral
Juxtacanalicular
Inner wall of Schlemm's canal
Schwalbe's line
Aqueous humor flow
Canal of Schlemm
Scleral spur
Iris

Fig. 6.5.2: Structure of trabecular meshwork

- Schlemm's canal
  - It is a circular canal that is present underneath the blue zone of the limbus circumferentially
  - On the cross-section, it is oval and is lined by endothelium
  - The aqueous is thought to enter the canal by vacuolation into the endothelial cells
- Collector channels
  - About 25–35 in number
  - Larger vessels run a short intrascleral course and empty into episcleral veins (direct system)
  - Smaller collector channels form an intrascleral plexus and drain into the episcleral veins (indirect system)

2. **Uveosceleral Pathway**
   - Anterior chamber → Suprachoroidal space → Veins of choroid and sclera (10% drainage)

2. A. Identify the instrument and state its use. (2 marks)
B. Explain Shaffer's grading with suitable drawings. (5 marks)
C. Describe three abnormalities that can be detected during this procedure. (3 marks)



A. The Instrument Shown is

- Gonio mirror
- It is used to measure the angle of the anterior chamber

B. Shaffer's Grading (Fig. 6.5.3)

Grade 0	No angle structures visible	Closed	0°
Grade I	Schwalbe's line	High chance of angle-closure- very narrow-angle	10°
Grade II	Schwalbe's line, trabecular meshwork	Angle-closure possible- narrow-angle	20°
Grade III	Schwalbe's line, trabecular meshwork, scleral spur	Open-angle-no chance of angle-closure	20–35°
Grade IV	Schwalbe's line, trabecular meshwork, scleral spur, ciliary body band	Wide-open angle-no chance of angle-closure	35° –45°

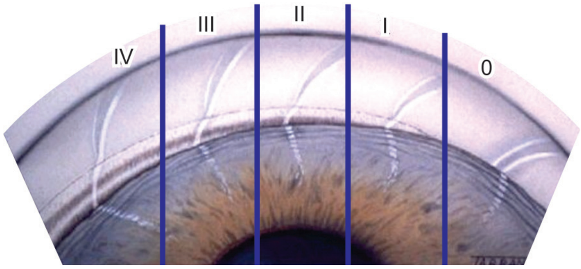


Fig 6.5.3: Grading of the angle

C. Abnormalities Detected on Gonioscopy vis-a-vis Causes/Associations

Abnormalities on Gonioscopy	Causes
• Peripheral anterior synechiae: ▪ Adhesions between iris and cornea seen as broad patchy adhesions bridging the angle between iris and Schwalbe's line	• Primary angle-closure glaucoma • Iridocyclitis • Iridocorneal endothelial syndrome
• Increased pigmentation along Schwalbe's line	• Pigmentary glaucoma • Pseudoexfoliation syndrome • Aphakic glaucoma • Blunt trauma • Anterior uveitis • Iris melanoma • Following Nd YAG iridotomy • Nevus of Ota
• Widened ciliary body band	• Angle recession glaucoma
• Neovascularization in the angle	• Neovascular glaucoma • Fuch's heterochromic iridocyclitis
• Blood in Schlemm's canal	• Increased episcleral venous pressure • Trauma • Caroticocavernous fistula

3. A. Identify the instrument and procedure. (2 marks)
B. Define intraocular pressure and give the normal range. (2 marks)
C. Describe diurnal variation of intraocular pressure and its implications. (3 marks)
D. Enumerate the factors that influence IOP dynamics. (3 marks)

A. Identify the Instrument

The Instrument Shown Is

Schiotz tonometer

Procedure is Called

Tonometry (the measurement of intraocular pressure)

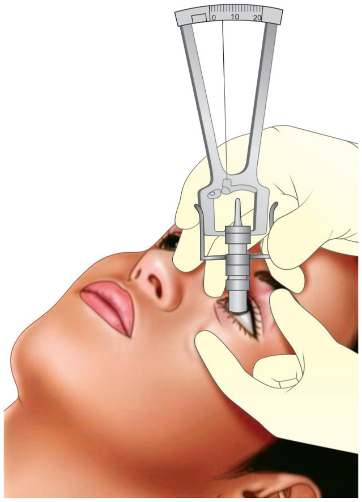
B. Intraocular Pressure

Definition

The pressure exerted by the contents of the eye on the coats of the eyeball

Normal Range

10.5–20.5 mm Hg



C. Diurnal Variation of IOP

- IOP fluctuates cyclically throughout the day (Fig. 6.5.4)
- The intraocular pressure is highest in the early morning and lowest in the evening. It follows a biphasic curve
- The normal difference between the highest and lowest values should be always less than 4 mm Hg
- If the difference is more than 4 mm Hg, it is suspicious of glaucoma and >8 mm Hg is pathognomonic of POAG

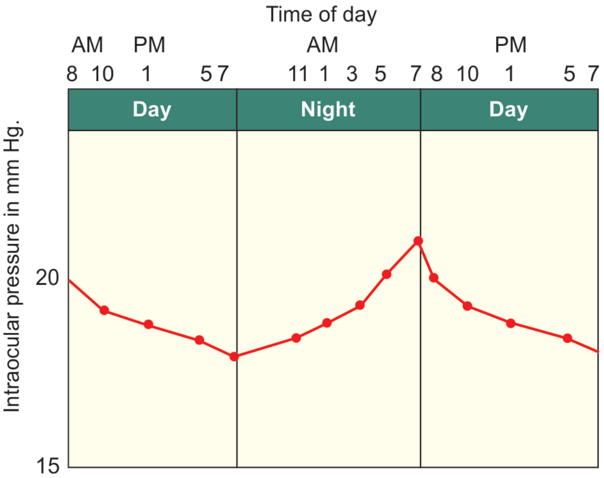


Fig. 6.5.4: Diurnal variation of IOP

Implications

1. Charting of IOP every 2–4 hours for 24 hours is needed to know the diurnal variation of IOP. It is known as phasing
2. Phasing is an important requirement for the diagnosis of POAG when the initial IOP may not be higher than 21 mm Hg
3. Phasing helps to know the highest pressure, so the target pressure can be set accordingly

D. Factors Affecting IOP Dynamics

1. **Age**
   - IOP increases with age, probably due to decreased drainage
2. **Heredity**
   - Many genes have been linked to causing different types of glaucoma
   - They interfere with the drainage of the aqueous
3. **Sleep**
   - IOP decreases during sleep

4. **Diurnal Variation**
    - IOP tends to peak early in the morning
    - This is the basis for phasing of IOP (every 4 hours) in a glaucoma suspect patient to find the peak
    - Normal eyes have a smaller fluctuation (<5 mm of Hg) than glaucomatous eyes (> 8 mm of Hg)
    - A biphasic curve, with two peaks, can be seen in primary open-angle glaucoma patients
5. **Postural variations**
    - IOP increases when changing from the sitting to the supine position
6. **Blood pressure**
    - It is not directly proportional to IOP, but some hypertensives who consume antihypertensive medications at night are found to develop normal-tension glaucoma
7. **Osmotic pressure of blood**
    - Increase in plasma osmolarity (like intravenous mannitol, oral glycerol, or in patients with uremia) can lead to a fall in IOP; whereas a reduction in plasma osmolality (as occurs with water drinking provocative tests) is associated with a rise in IOP
8. **General anesthetics and other drugs**
    - Ketamine is known to increase IOP

SHORT ESSAYS

1. Explain the structure of the angle of the anterior chamber. (5 marks)

- The angle of the anterior chamber is the space between the peripheral cornea and the root of the iris
- It is the site of aqueous drainage
- It consists of the following structures from posterior to anterior

Structure	Description
Root of the iris	• It is the posterior-most structure seen on gonioscopy • It is formed by the base of the iris and appears dark brown
Ciliary body band	• It is formed by the anterior-most part of the ciliary body • Its width depends on the iris insertion • It is wider in myopes and narrower in hypermetropes
Scleral spur	• It appears as a prominent white line • The longitudinal muscle of the ciliary muscle is attached to the posterior aspect of the scleral spur • Anteriorly, it is attached to the trabecular meshwork
Trabecular meshwork	• It is seen as a greyish-black band of varied pigmentation • It has three microscopic parts: Uveoscleral, corneoscleral, and juxtacanalicular • It is made of pores of decreasing sizes that act as a filter and help in the conventional aqueous outflow
Schwalbe's line	• It is a fine glistening ridge seen as the anterior-most structure of the angle of the anterior chamber • It is the prominent end of the Descemet's membrane of the cornea

2. Describe the structure and functions of the blood-aqueous barrier. (5 marks)

Blood-aqueous Barrier

Structure

- A blood-aqueous barrier is formed by the tight junctions between the cells of the inner nonpigmented epithelium of the ciliary body
- The tight junctions are zona occludens and zona adherens (Fig. 6.5.5)
- It is also formed by the nonfenestrated endothelium of the iris capillaries
- These are not joined by tight junctions and so, they lead to formations of cells and flare during inflammation

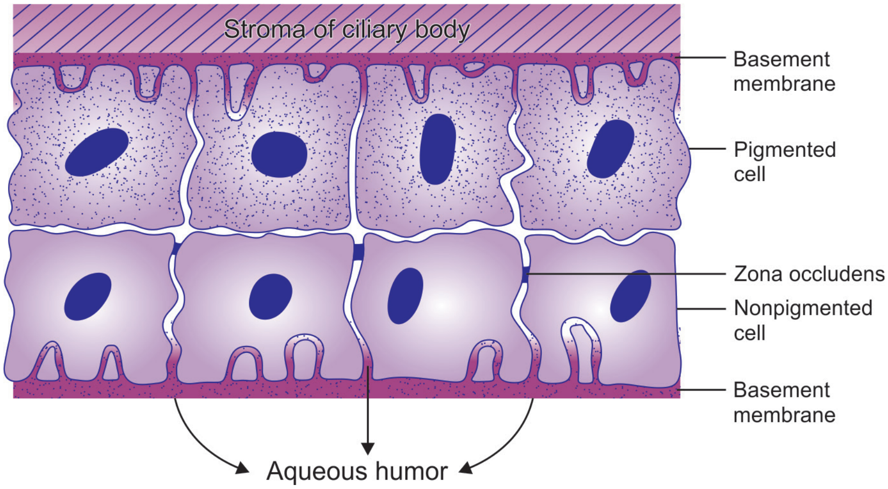


Fig. 6.5.5: Structure of blood-aqueous barrier

Functions

1. The blood-aqueous barrier prevents the entry of molecules into the aqueous and maintains its transparency and osmolarity
2. It is responsible for maintaining the difference in the chemical composition of the aqueous and plasma. An increase in contents of the aqueous (as in uveitis) causes the aqueous to become 'plasmoid' and thus makes the aqueous turbid
3. Low molecular weight molecules like chloramphenicol, cephalothin can penetrate the blood-aqueous barrier. Lipid soluble molecules pass easily. Thus, it helps in providing medications into the aqueous

SHORT ANSWERS

1. Explain the functions of aqueous humor. (3 marks)

1. Aqueous humor helps to maintain the IOP even though it occupies a smaller volume compared to the vitreous. This is because of its dynamic nature. IOP helps in maintaining the structure of the eyeball and helps in axoplasmic transport across the optic nerve head
2. It plays role in nourishing endothelium and lens
3. Helps to provide substrates and removing metabolic end products
4. Aqueous also provides an optically clear medium for vision
5. It has high ascorbate levels that prevent UV-induced oxidative damage
6. Facilitates cellular and humoral immunity responses

2. Contrast trabecular and uveoscleral outflow of aqueous. (3 marks)

Trabecular and Uveoscleral Outflow of Aqueous

Feature	Trabecular/conventional pathway	Uveoscleral pathway
Percentage of aqueous drainage	90%	10%
Pathway	Trabecular meshwork → Schlemm's canal → collector channels → aqueous veins → episcleral veins	anterior chamber → suprachoroidal space → veins of choroid and sclera
Factors affecting outflow	Dependent on IOP	Independent of IOP (not below 4 mm Hg) Age-dependent decrease in outflow Cyclodialysis causes an increased outflow
Process of aqueous outflow	Contraction of the ciliary body causes a pull on the scleral spur that pulls the TM and increases the size of the pores.	Relaxation of ciliary muscle causes an increase in outflow
Effect of antiglaucoma drugs that act	Beta-blockers Cholinergic Carbonic anhydrase inhibitors	Prostaglandins Alpha agonists Cholinergic drugs decrease aqueous outflow

COMPETENCY

OP6.6 IDENTIFY AND DEMONSTRATE THE CLINICAL FEATURES AND DISTINGUISH AND DIAGNOSE COMMON CLINICAL CONDITIONS AFFECTING THE ANTERIOR CHAMBER

SHORT ANSWER

1. Demonstrate the understanding of the procedure of gonioscopy and structures seen during this procedure. (3 marks)

Procedure of Gonioscopy

- The patient is seated at the slit-lamp. Topical anesthesia is instilled in both eyes
- A drop of methylcellulose is placed in the concavity of the lens
- The beam height is decreased to 3 mm so that it does not cross the pupil
- The gonio-lens is placed on the cornea and the slit beam is focused on the inferior mirror. This shows the angle structures of the superior angle
- This is followed by an examination of each quadrant of the angle

Structures Seen in Gonioscopy

Root of the iris	• It is the posterior-most structure seen on the gonioscopy • It is formed by the base of the iris and appears dark brown
Ciliary body band	• It is formed by the anterior-most part of the ciliary body • Its width depends on the iris insertion • It is wider in myopes and narrower in hypermetropes
Scleral spur	• It appears as a prominent white line • The longitudinal muscle of the ciliary muscle is attached to the posterior aspect of the scleral spur • Anteriorly, it is attached to the trabecular meshwork

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Trabecular meshwork	• It is seen as a grayish-black band of varied pigmentation • It has three microscopic parts: Uveoscleral, corneoscleral, and juxtacanalicular • It is made of pores of decreasing sizes that act as a filter and help in the conventional aqueous outflow
Schwalbe's line	• It is a fine glistening ridge seen as the anterior-most structure of the angle of the anterior chamber • It is the prominent end of the descemet membrane of the cornea

COMPETENCY

OP6.7 ENUMERATE AND DISCUSS THE AETIOLOGY, THE CLINICAL DISTINGUISHING FEATURES OF VARIOUS GLAUCOMAS ASSOCIATED WITH SHALLOW AND DEEP ANTERIOR CHAMBER. CHOOSE APPROPRIATE INVESTIGATIONS AND TREATMENT FOR PATIENTS WITH ABOVE CONDITIONS

LONG ESSAY

1. A 45-year-old man came for a regular eye check-up. His visual acuity was 6/6, N6 in both eyes after correcting presbyopia. The intraocular pressure was 30 mm Hg and 28 mm Hg in both eyes, and the discs showed an increase in the size of cupping.

A. What is the most probable diagnosis? (1 mark)
B. How is it confirmed? (1 mark)
C. Explain the disc changes associated with this condition. (4 marks)
D. Explain any 4 visual field changes associated with this condition. (4 marks)
E. Enumerate the classes of drugs useful in treating this condition with one example for each. (3 marks)
F. Describe ocular side effects of latanoprost. (2 marks)

A. Most Probable Diagnosis

Primary open-angle glaucoma

B. Diagnosis is Confirmed by

An automated visual field analysis

C. Disc Changes Associated with Glaucoma

Early Glaucomatous Changes

1. **Notching of neuroretinal rim (Fig. 6.7.1)**
   - Vertically oval cup due to selective loss of neural rim tissue in the inferior and superior poles. (ISNT rule → order of loss of neuroretinal rim: inferior > superior > nasal > temporal
2. **Asymmetry of the cups (Fig. 6.7.2)**
   - Adifference of more than 0.2 between two eyes is significant
   - Large cup i.e., 0.6 or more
   - Even if symmetrical, a cup more than 0.6 is suspicious

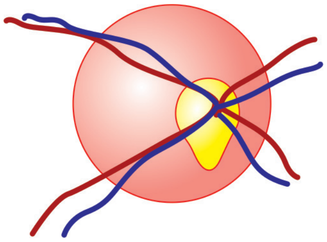


Fig. 6.7.1: Notching of neuroretinal rim

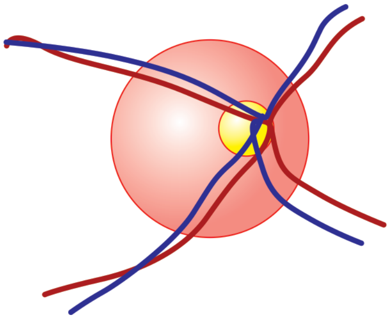


Fig. 6.7.2: Asymmetric cup

3. **Baring of circumlinear vessel (Fig. 6.7.3)**
    - A circumlinear vessel traverses along the rim of the cup
    - When the cup recedes, the vessels seem 'bared' or left afloat
4. **Splinter hemorrhages (Drance hemorrhages) (Fig. 6.7.4)**
    - Present on or near the optic disc margin, usually represent a sudden increase in glaucomatous damage
5. **Thinning of retinal nerve fiber layer (RNFL loss)**
    - May be seen as fan-shaped areas of thinning starting from the disc, especially in the inferonasal part
    - It is appreciated better with red-free light

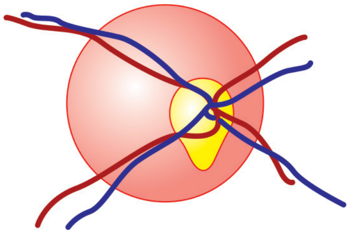


Fig. 6.7.3: Baring of circumlinear vessel

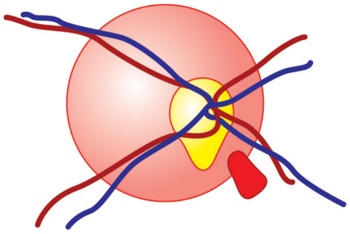


Fig. 6.7.4: Disc hemorrhage

Advanced Glaucomatous Changes

1. **Marked cupping**
    - (Cup size 0.7 to 0.9), excavation may even reach the disc margin (total loss of neuroretinal rim), the sides are steep (Fig. 6.7.5)
2. **Laminar dot sign (Fig. 6.7.6)**
    - Due to extreme thinning of the nerve layer, underlying lamina cribrosa is visible on ophthalmoscopy
    - Nasalization of vessels: The cup is usually a bit nasal. When there is progressive cupping, the cup extends temporally whereas the vessels remain. Hence, it appears like vessels are shifted nasally
3. **Bayoneting sign (Fig. 6.7.7)**
    - Double angling of vessels as they pass over the deep cup.

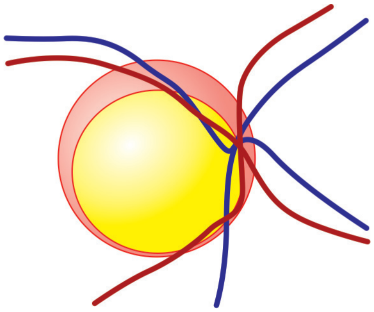


Fig. 6.7.5: Advanced cupping

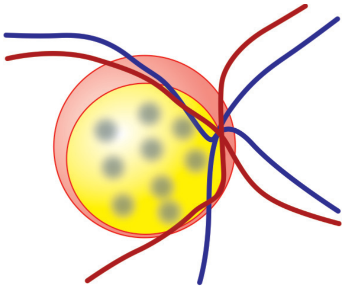


Fig. 6.7.6: Laminar dot sign

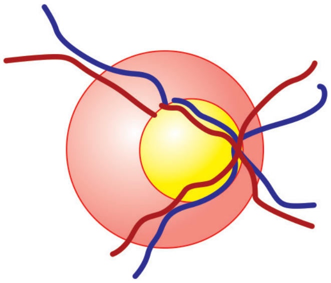
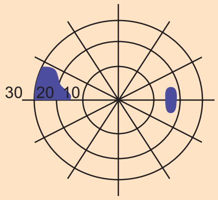
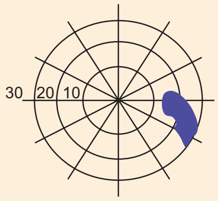
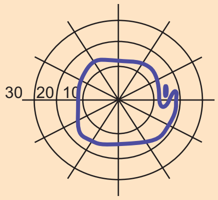


Fig. 6.7.7: Bayonetting sign

4. **Peripapillary atrophy**
   - Due to increased pressure, there is ischemia due to compression of short posterior choroidal vessels, which supply the peripapillary choroid
5. **Glaucomatous optic atrophy**
   - It is seen as a part of end-stage glaucoma, characterized by pale disc, advanced cupping, and other signs of advanced glaucoma

D. Visual Field Changes Associated with Glaucoma

Name of field defect	Description	Example
Nasal step (Fig. 6.7.8)	Can occur in the periphery (shown in picture), mid periphery (tends to perpendicular), or near the fixation (obtuse angle with raphe)	 **Fig. 6.7.8** Bayonetting sign
Temporal wedge (Fig 6.7.9)	Can be supero- or inferonasal; extension from the temporal edge of blind spot	 **Fig. 6.7.9** Temporal wedge
Baring of blind spot (Fig. 6.7.10)	It occurs as a result of an increased sensitivity of the retina below the blind spot compared to the same area above the blind spot	 **Fig. 6.7.10** Baring of blind spot

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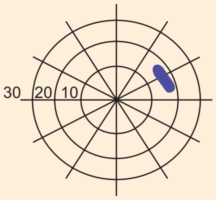
Name of field defect	Description	Example
Paracentral scotoma (Fig. 6.7.11)	Usually seen as a relative steep scotoma occurring in the superonasal quadrant	 **Fig. 6.7.11** Paracentral scotoma

E. Drugs Used in Open-angle Glaucoma

1. **Beta blockers:** Timolol
2. **Prostaglandin analogs:** Latanoprost
3. **Alpha agonists:** Brimonidine
4. **Carbonic anhydrase inhibitors:** Dorzolamide
5. **Cholinergic drugs:** Pilocarpine

F. Ocular Side Effects of Latanoprost

1. Conjunctival hyperemia, foreign body sensation, eye irritation and superficial punctuate keratopathy-temporary side effects. It is a quite common side effect seen with Latanoprost
2. Itching, burning sensation, and eye pain
3. Reduced corneal sensation, thickness
4. Periocular hyperpigmentation has been noted in most patients on long-standing treatment, which tends to disappear on discontinuation of the drug
5. Increase in length and number of lashes, long enough that can rub against the spectacles
6. Activation of latent Herpes and uveitis is also a complication
7. Cystoid macular edema along with uveitis which responds to NSAIDs
8. Darkening of iris
9. Choroidal effusion

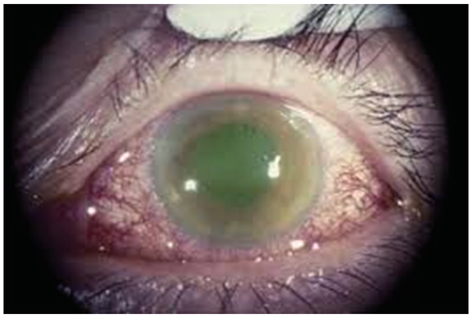
SHORT ESSAYS

1. A 50-year-old woman presents with acute onset redness, pain, and diminution of vision in the right eye. She gave the history of the appearance of colored halos on and off for the last six months. Her anterior segment shows the following image.

A. What is the most probable diagnosis? (1 mark)

B. What is the probable finding if gonioscopy is performed in this stage? (1 mark)

C. Explain the management of this condition. (3 marks)



A. Most Probable Diagnosis

Right eye acute angle-closure glaucoma or congestive attack of angle-closure glaucoma.

B. If Gonioscopy is Performed

The angle is closed or grade 0. No structures are visible on the gonioscopy.

C. Management

1. **Immediate management**
   - Make the patient lie down supine (So that lens shift posteriorly)
   - Intravenous or oral acetazolamide 500 mg (To decrease aqueous production)
   - Topical steroid (dexamethasone or prednisolone) QID (To decrease the inflammation)
   - Topical timolol 0.5%
   - Analgesics and anti emetics
   - Draw blood for baseline sugars, electrolytes, and renal function tests
2. **After one hour**
   - Topical pilocarpine 2%, in both eyes, QID (It acts by straightening the iris and decreasing the contact between the anterior surface of the lens and the posterior surface of the iris.)
   - After 30 more minutes
   - Recheck IOP—if not fallen <35 mm Hg, oral or intravenous hyperosmotic drugs, IV 20% mannitol 1g/kg over 45–60 min (1 drop per second) or oral 50% glycerol 1g/kg
3. **Definitive management**
   - Perform gonioscopy to understand the synechiae status of the eye
   - Laser peripheral iridotomy in the affected eye, when IOP has dropped and is around 20–25 mm Hg. (The principle is to create a bypass for the flow of aqueous in the case of pupillary block)
   - Laser peripheral iridotomy in the fellow eye

2. Define glaucoma. Classify glaucoma based on gonioscopy. (2+3 marks)

Definition of Glaucoma

- A chronic, progressive optic neuropathy associated with damage of the optic nerve head associated with characteristic or typical disc and visual field changes, usually associated with an increase in IOP
- Based on gonioscopy, glaucoma can be broadly classified as open-angle and closed-angle glaucoma
- An angle is said to be open if trabecular meshwork is visualized during gonioscopy

Classiifcation of Glaucoma Based on Gonioscopy

I. Open-angle Glaucoma

1. **Primary open-angle glaucoma**
   - i. Normal-tension glaucoma
   - ii. Ocular hypertension
2. **Secondary open-angle glaucoma**
   - i. Pseudo-exfoliation glaucoma
   - ii. Pigmentary glaucoma
   - iii. Aphakic glaucoma
   - iv. Steroid-induced glaucoma
   - v. Uveitic glaucoma

II. Angle-closure Glucoma

1. **Primary angle-closure glaucoma**
    i. Primary angle-closure suspect
    ii. Primary angle-closure
    iii. Primary angle-closure glaucoma
2. **Secondary angle-closure glaucoma**
    i. Neovascular glaucoma
    ii. Lens induced glaucoma
    iii. Uveitic glaucoma

3. Define the following terms (1 mark each)

A. Primary open-angle glaucoma
B. Ocular hypertension
C. Normal-tension glaucoma
D. Primary angle-closure glaucoma
E. Juvenile glaucoma

A. Primary Open-angle Glaucoma

When the intraocular pressure is more than 21 mm Hg, associated with glaucomatous disc changes and visual field changes, in the presence of an open angle and without any secondary ocular cause.

B. Ocular Hypertension

It is defined when the intraocular pressure is more than 21 mm Hg with no optic disc changes or visual field changes in the presence of an open-angle.

C. Normal-tension Glaucoma

It is defined when the intraocular pressure never exceeds 21 mm Hg, but the eyes show glaucomatous optic disc and visual field changes in the presence of an open-angle.

D. Primary Angle-closure Glaucoma

It is defined when the intraocular pressure is more than 21 mm Hg, associated with glaucomatous disc changes and visual field changes, in the presence of a narrow or closed-angle and without any secondary ocular cause.

E. Juvenile Glaucoma

Presence of raised intraocular pressure with characteristic disc and visual field changes in presence of an open angle and no other ocular associations seen in the age group of 10–35 years.

4. Define the visual field. Explain the normal visual field with the neat and labeled diagram and its values in four quadrants. (1+4 marks)

Definition of Visual Field

It is the area of space that is seen by an eye when fixated onto a point within that space.

Normal Visual Filed

- Normally, it is widest temporally (90–100°), followed by inferiorly (70–80°), superiorly (60°), and least nasally (55–60°) (Fig. 6.7.12).

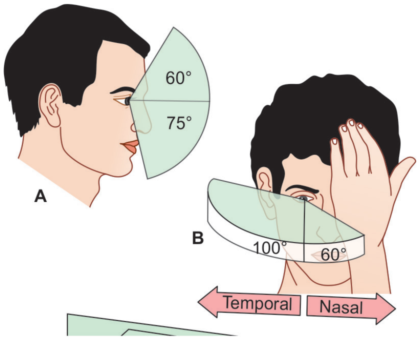


Fig. 6.7.12: Normal visual field

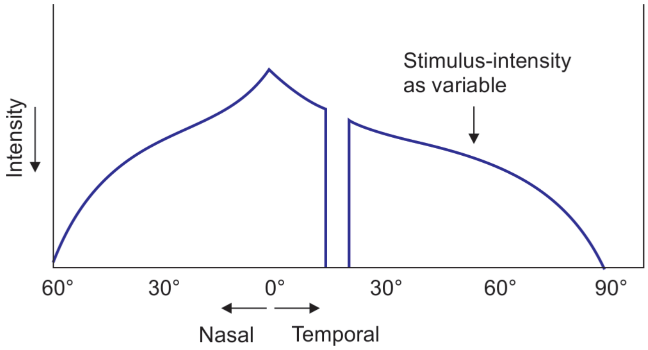
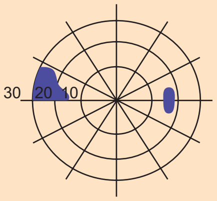
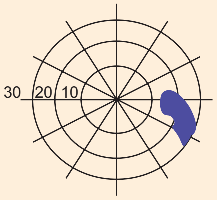
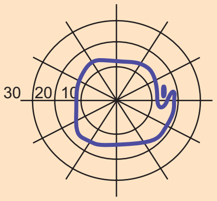


Fig. 6.7.13: Hill of vision

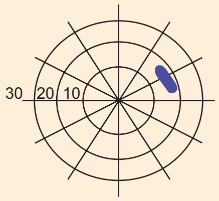
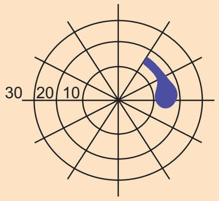
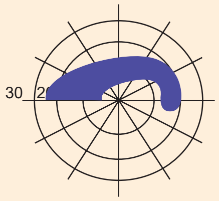
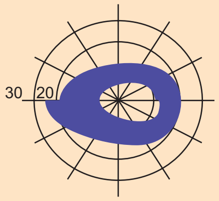
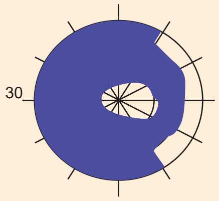
- The sensitivity of the retina peaks at the macula with a ''bottomless trough'' at the physiological blind spot (entrance of optic nerve) (Fig 6.7.13)
- The blind spot lies at the nasal side in the retina and hence is perceived temporal to fixation at approximately 12–15°
- The automated perimeter measures the sensitivity at all points on the retina in various quadrants by presenting to the patient, stimuli of varying size and brightness

5. Describe the visual field changes in glaucoma (5 marks)

Visual Field Changes Associated with Glaucoma

Name of field defect	Description	Example
Nasal step (Fig. 6.7.14)	Can occur in the periphery (shown in picture), mid periphery (tends to perpendicular), or near the fixation (obtuse angle with raphe)	 **Fig. 6.7.14:** Bayonetting sign
Temporal wedge (Fig. 6.7.15)	Can be supero- or inferonasal; extension from the temporal edge of blind spot	 **Fig. 6.7.15:** Temporal wedge
Baring of blind spot (Fig. 6.7.16)	It occurs as a result of an increased sensitivity of the retina below the blind spot compared to the same area above the blind spot	 **Fig. 6.7.16** Baring of blind spot

Contd.

Name of field defect	Description	Example
Paracentral scotoma (Fig. 6.7.17)	Usually seen as a relative steep scotoma occurring in the superonasal quadrant	 **Fig. 6.7.17** Paracentral scotoma
Seidel's scotoma (Fig. 6.7.18)	Paracentral scotoma extends into the blind spot to form a Seidel scotoma	 **Fig. 6.7.18:** Seidel scotoma
Arcuate scotoma (Fig. 6.7.19)	Seidel scotoma extends into the temporal half to reach the horizontal raphe to form an arc Can be superior or inferior	 **Fig. 6.7.19:** Arcuate scotoma
Ring scotoma or double arcuate scotoma (Fig. 6.7.20)	When an inferior and a superior arcuate join at the horizontal raphe, a discrepancy is seen as a sharp perpendicular step. It is called Roenne's nasal step	 **Fig. 6.7.20:** Ring scotoma
Tunnel vision (Fig. 6.7.21)	A central tubular vision is remaining along with a peripheral temporal crescent.	 **Fig. 6.7.21:** Tubular vision
Isopteric contraction	Decrease in the central or peripheral viewing area	

6. Define perimetry. Describe the procedure of Static perimetry and its use in glaucoma. (1+2+2 marks)

Definition of Perimetry

The process by visual field is charted is called perimetry.

Procedure of Static Perimetry

- In static perimetry, stimuli of varying brightness are presented at various points in the visual field, and the patient is asked to press a switch upon seeing the stimulus
- Here, sensitivity is determined at each point on the retina and is measured in terms of apostilbs (abs)
- It is done using an automated perimeter called Humphrey's perimeter
- It has a dome-shaped bowl with central fixation and it has computerized software that calculates the sensitivity of the retina at each point
- The patient is seated at the mouth of the bowl with one eye patched and chin placed at the chin rest
- Appropriate refractive correction is placed
- The patient is given a button that is connected to the system
- The patient is instructed to click the button upon seeing a spot of light
- The computer offers many points in the visual field that is to be tested (entered into the computer by the operator). The field usually chosen for glaucoma is 30–2 or 24–2
- Depending on the strength of the stimulus presented at different positions in the bowl and the patient's response. A printout of the test result is provided after the test

Use in Glaucoma

1. The foremost use of perimetry in glaucoma is to establish a diagnosis of glaucoma
    - The common visual field changes seen in glaucoma are baring of the blind spot, siedel scotoma, Arcuate scotoma, and annular scotoma
2. Diagnosis of normal-tension glaucoma is made when there is a worsening of visual field defect
    - In presence of normal intraocular pressure and a stationary field defect, a diagnosis of normal-tension glaucoma cannot be made
3. The next use in understanding the progression of glaucoma and the efficacy of the treatment provided
    - The Humphrey perimeter house software helps to understand the progression of glaucoma based on three consecutive field tests
    - It can also be done manually by comparing the mean deviation, pattern standard deviation, and understanding the greyscale

7. Describe the etiology, clinical features, and management of primary congenital glaucoma. (1+2+2 marks)

Primary Congenital Glaucoma

A distinct entity, characterized by anatomical disturbances in the drainage of aqueous humor due to goniodysgenesis

Etiology

- It is sporadic in occurrence with a very high male preponderance
- It can be hereditary with polygenic inheritance

- The embryological basis for PCG is the cessation of the iris root and ciliary body recede posteriorly during the development of the angle
- As a result, the iris is inserted anteriorly into the angle
- The three major abnormalities seen are:
  1. Isolated trabeculodysgenesis
  2. Iridodysgenesis
  3. Corneodysgenesis

Clinical Features

- The presentation of childhood glaucomas is unique. Parents bring the child with excess tearing, child burying its head into pillow and blepharospasm. It is due to irritation of corneal nerves due to corneal edema
- The cornea appears hazy due to edema and is enlarged (buphthalmos—bull eye)
- Normally, the diameter of the cornea is 10.5 mm; more than 13 mm is considered pathognomonic
- Haab's striae: Tears in Descemet's membrane are seen as parallel peripheral concentric tears
- The sclera is thinned due to increased pressure and appears blue
- The anterior chamber is deep
- Iris appears thinned and the lens is stretched
- The optic disc shows cupping or atrophy due to increased pressure
- Axial myopia is seen

Management

- Definitive treatment is always surgical
- A short period of medical therapy with topical beta-blockers or carbonic anhydrase can be given to control IOP in case the child is unfit for surgery or before surgery
- The procedures that can be performed are:

1. **Goniotomy**
   - In this procedure, a Barkan's knife is introduced into the anterior chamber under gonioscopic control and advanced to the opposite angle and an incision is made in the midway of the angle and the knife is rotated for 75°
   - A similar procedure can be done on the other side at the next sitting
2. **Trabeculotomy**
   - It is the next step if goniotomy fails
   - It is also indicated when cloudy or scarred cornea permits the view of angle by gonioscopy
   - A small vertical incision is made on the sclera at the level of the Schlemm's canal at 12'o clock position
   - One prong of the trabeculotome is passed into the Schlemm's canal circumferentially for 90°
   - The upper prong acts as a guide
3. **Trabeculectomy**
   - It is a controlled aqueous filtration procedure in which a window is made in the trabecular meshwork and aqueous is made to filter out of the anterior chamber into the subconjunctival space
4. **Glaucoma drainage devices**
   - Synthetic devices are placed surgically in the subconjunctival space
   - It is indicated if the above procedures fail

5. **Cyclodestructive procedures**
   - It is indicated in intractable glaucoma with no visual potential; where a part or whole of the ciliary body is destroyed either by cryoapplication or LASER to decrease aqueous production

8. A 3-month-old child is brought with bluish appearing, enlarged corneas. The parents give a history of the child burying her head in pillows and the presence of watering.

A. Enumerate three causes of epiphora in a neonate and differentiate among the three. (3 marks)

B. What is the probable condition the child is suffering from? (1 mark)

C. Give one ocular association. 1 mark)

A. Causes of Epiphora in a Neonate

1. Congenital dacryocystitis
2. Ophthalmia neonatorum
3. Primary congenital glaucoma

Differentiation

Parameter	Congenital dacryocystitis	Ophthalmia neonatorum	Primary congenital glaucoma
History	Watering and occasional discharge Starts within the first month	Watering with or without discharge may start as early as the first two days of birth	Can manifest anytime before three years of age. Signs of photophobia (buries head in pillow)
Family history	Not significant	Genital herpes or gonorrhea in the mother may be important	Not relevant
Laterality	Usually unilateral	Unilateral or bilateral	Bilateral
Cornea	Normal	May be affected in cases of keratitis	Enlarged and hazy (buphthalmos)
Sclera	Normal	Normal	Thinned
Axial length	Normal	Normal	High
Intraocular pressure	Normal	Normal	High
Regurgitation test	Positive	Normal	Normal
Lacrimal syringing	Obstruction	Normal	Normal

B. Most Probable Diagnosis

Primary congenital glaucoma

C. Ocular Association

Axenfeld anomaly

9. Explain the risk factors for the development of primary open-angle glaucoma. Describe the role of steroid responsiveness in primary open-angle glaucoma. (3+2 marks)

- Primary open-angle glaucoma is a condition with no underlying ocular associations
- The genetic basis of POAG is linked to the genome GLC-1A. two main genes are involved—myocilin (chromosome 1q21-31) and optineurin (10p)

Risk Factors for the Development of Primary Open-angle Glaucoma

1. **Heredity:** Inheritance is polygenic, a familial tendency exists. If a person develops POAG before 35 years, there is a high chance his siblings will develop it too due to gene mutation
2. **Age:** Risk increases with an increase in age. It is seen in the 6th and 7th decade onwards
3. **Myopes:** They have more chances of developing POAG
4. Diabetics have a higher prevalence of glaucoma
5. Ocular risk factors include thin central cornea (due to associated thin lamina cribrosa), vertical cup and reduced diastolic perfusion pressure (diastolic blood pressure-intraocular pressure)

Steroid Responsiveness

- A large subset of POAG patients are high responders (develop a pressure of more than 30 mm Hg when treated with topical dexamethasone for 6 weeks)
- Steroids induced production of myocilin in the trabecular meshwork leading to decreased outflow
- It is therefore understood that steroid responsiveness is genetically inherited and very closely linked to POAG
- Hence, steroids have to be carefully used in patients with POAG

11. Describe the following drugs concerning concentration, dose, indications, and two side effects.

A. Pilocarpine (2.5 marks)

B. Timolol (2.5 marks)

Drug	Concentration	Dose	Indications	Side effects
Pilocarpine	1%, 2%, 5%, 10%	QID	• Primary angle closure glaucoma • Plateau iris syndrome • Before performing LASER peripheral iridotomy	• Conjunctival injection, ocular and periocular pain (due to ciliary spasm), twitching of eyelids • Fluctuating myopic shift of up to 12–15D
Timolol	0.25%, 0.5% (higher percentage used in darker irides)	BID	• Primary open-angle glaucoma • Steroid-induced glaucoma • Preoperatively to control IOP in congenital glaucoma. • Glaucoma is associated with uveitis. • Glaucoma associated with hyphema • Angle-closure attack: to decrease IOP so that pilocarpine can act	• Can induce bronchospasm: to be carefully used in asthmatic patients. • Can decrease heart rate and BP: Great caution in patients with heart failure, sinus bradycardia, hypotension, hypokalemia, and brittle diabetes.

11. Explain the ocular and systemic side effects of topical beta-blockers used for glaucoma. (2+3 marks)

Ocular Side Effects

1. Stinging, burning, or itching
2. Timolol can exacerbate keratoconjunctivitis sicca, can cause periocular dermatitis, allergic conjunctivitis, and punctate keratitis
3. Long-term drift-using for long-duration causes decreased sensitivity to the drug

Systemic Side Effects

1. Can induce bronchospasm: To be carefully used in asthmatic patients
2. Can decrease heart rate and BP: Great caution in patients with heart failure, sinus bradycardia, hypotension, hypokalemia, and brittle diabetes
3. Symptoms related to the central nervous system are common in patients receiving β blocker therapy. They are mood swings, fatigue, confusion, depression, and loss of concentrating ability
4. Timolol can cause slowing of fetal heart rate and ventricular arrhythmias. Hence β blockers have to be used with extreme caution in pregnant women
5. Long-term use of timolol has been found to cause an increase in triglycerides and low-density lipoproteins carteolol has a slightly better therapeutic index in such patients than does timolol

12. Describe the mechanism of action and adverse effects of prostaglandins as antiglaucoma medications. (2+3 Marks)

Prostaglandins as Antiglaucoma Medications

Mechanism of Action

- They have mixed pharmacological responses because of the diversity of receptors
- The prostaglandin receptors are widely distributed in the eye accounting for their diverse biological effects
- Prostaglandin analogs combine with FP receptors and cause
  1. Relaxation of the ciliary muscle increases the aqueous outflow by the uveoscleral pathway
  2. They bring about the remodeling of the extracellular matrix by induction of the nuclear transcription factor and c-FOS. This causes the release of matrix metalloproteinases and that brings about degradation of the collagen, fibronectin in the ciliary muscle, and trabecular meshwork
  3. Other mechanisms for prostaglandin are proposed: Increase in blood flow to the optic nerve head and interaction with adrenergic pathway and increase in the conventional pathway by relaxation of the contractile trabecular meshwork fibers

Adverse Effects

- Prostaglandins have more local side effects
  1. Conjunctival hyperemia, foreign body sensation, eye irritation and superficial punctuate keratopathy-temporary side effects
  2. Itching, burning sensation, and eye pain
  3. Reduced corneal sensation, thickness

    4. Periocular hyperpigmentation has been noted in most patients on long-standing treatment, which tends to disappear on discontinuation of the drug
    5. Increase in length and number of lashes, long enough that can rub against the spectacles
    6. Activation of latent herpes and uveitis is also a complication
    7. Cystoid macular edema along with uveitis which responds to NSAIDs
    8. Darkening of iris
    9. Choroidal effusion
- Systemic side effects are minimal, except for headache and muscle pains
- Prostaglandins are best avoided in pregnant women, especially in the last three trimesters for the fear of termination of pregnancy

13. Describe the mechanism of action and adverse effects of carbonic anhydrase inhibitors as antiglaucoma medications. (2+3 marks)

Carbonic Anhydrase Inhibitors as Antiglaucoma Medications

Mechanism of Action

- Carbonic anhydrase enzyme catalyzes the following reaction
  $CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow HCO_3 + H^+$
- This step is critical for the formation of aqueous. Carbonic anhydrase inhibitors block the transport of bicarbonate ions from inside to outside of the cell
- As a result, intracellular bicarbonate increases
- Chloride-bicarbonate channels are activated. As a result, the intracellular concentration of chloride increases causing an acidic environment
- This inhibits the net flux of chloride across the ciliary epithelium
- As a result, the net concentration of sodium, chloride, and bicarbonate decreases in the aqueous

Adverse Effects

- Ocular side effects
    1. Irritation
    2. Blurring of vision
    3. Hypersensitivity reactions
- Systemic side effects
    1. Paresthesia around mouth and fingertips
    2. Gastritis, nausea, diarrhea
    3. Hypokalemia
    4. Metabolic acidosis—avoid hepatic insufficiency, renal insufficiency, severe pulmonary obstruction, and hyperchloremic acidosis
    5. Stevens–Johnson syndrome
    6. Thrombocytopenia, agranulocytosis, aplastic anemia

14. Describe cyclodestructive procedures. Give one indication and one side effect. (3+1+1 marks)

Cyclodestructive Procedures

- These are glaucoma surgeries in which a part or whole of the secretory part of the ciliary body is destroyed by various mechanisms

- They are:
  1. **Cyclo-cryotherapy:** Cryotherapy is applied at the region of the ciliary body (3 mm from limbus) for a minute. This is done in an area of 180° circumferentially
  2. **Nd: YAG Laser cyclodestruction:** ND: YAG Laser is used to destroying the ciliary body
  3. **Endo-cyclophotocoagulation:** It uses Diode LASER, xenon, helium-neon aiming beam, and LASER is delivered through slit-lamp
  4. **Diode transscleral cyclophotocoagulation:** 810 nm LASER is used to destroy the ciliary epithelium under peribulbar anesthesia

Indication

Absolute glaucoma due to any cause

Side Effect

Hypotony

15. Describe the risk factors, clinical features, and management of normal-tension glaucoma. Give one differential diagnosis. (1+1+2+1 marks)

Normal-tension Glaucoma

A subset of POAG where the intraocular pressure is always lesser than 21 mm Hg but the eyes show characteristic optic disc and visual field changes in presence of an open-angle

Risk Factors

1. Old age
2. Use of antihypertensive drugs in the night (decreases the perfusion to the optic nerve head)
3. Vascular diseases like Raynaud's phenomenon and peripheral vascular disease, migraine

Clinical Features

- They are very similar to POAG, except that IOP is normal and always less than 21 mm Hg
- They too have a very large fluctuation of IOP
- Visual fields in the NTG subgroup tend to approach fixation more commonly than POAG

Management

- Reduction in IOP to low teens is desired to decrease progression
- Betaxolol is the drug of choice in NTG patients due to its beta1 specificity, and no associated vasoconstriction. It also has neuroprotective action
- Prostaglandin analogs are very effective in decreasing IOP
- Brimonidine is useful due to its neuroprotection
- Systemic calcium channel blockers in proven cases of vasospasm
- Trabeculectomy is considered in failure to medical treatment

Differential Diagnosis

Steroid induced glaucoma (after cessation of steroids).

16. Describe the arrangement of retinal nerve fiber at optic disc and explain the reason for polar notching as an early sign of glaucoma. (3+2 marks)

Arrangement of Retinal Nerve Fiber Set Optic Disc (Fig. 6.7.22)

1. **Nasal fibers (radially arranged)**
   - They are called superior radial and inferior radial fibers (SRF and IRF)
   - They represent the temporal field which is the largest of all. So, the nerve fibers are thicker
2. **Macular fibers (macula to optic disc-papillomacular bundle)**
   - These are the thinnest fibers and highly dependent on ATP and have more mitochondria
   - They are the last to swell during papilledema but the first to be affected in toxic amblyopia, Leber's hereditary optic neuropathy, and B12 deficiency, all of which leads to depletion of ATP
3. **Temporal fibers (arch around macular fibers)**
   - They are called superior and inferior arcuate fibers (SAF and IAF)
   - They represent the nasal visual field
   - So, when there is temporal retinal detachment, the nasal field is lost and vice versa

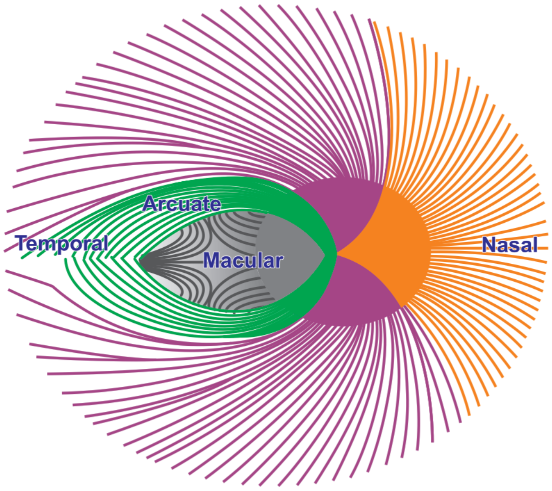


Fig. 6.7.22: Arrangement of nerve fibers

Polar Notching (Fig. 6.7.23)

- It is the phenomenon in which the neuroretinal rim in the upper and lower poles thin out as the earliest feature of glaucoma
- The cup starts to appear vertically oval
- This is because these fibers are more in number and get compressed easily with raised intraocular pressure or become vulnerable to hypoxia
- As a result, they are more sensitive to glaucomatous damage. So, polar notching is an early sign of glaucoma

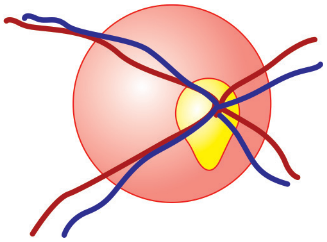


Fig. 6.7.23: Polar notching

17. Describe five investigations used in the diagnosis and follow-up of glaucoma. (5 marks)

Investigations Used In The Diagnosis and Follow-up of Glaucoma

1. **Tonometry and gonioscopy**
   - Tonometry is a measurement of IOP and is an important part of the examination for glaucoma diagnosis and monitoring treatment. It is best done with applanation tonometry or tonopen. A normal IOP is 10–21 mm Hg. Diurnal variation of more than 8 mm is diagnostic of glaucoma
   - Gonioscopy is required to classify glaucoma is open-angle or angle-closure. It is also useful to know other associated pathologies like neovascularization, synechiae, ICE syndromes, pigmentary glaucoma, and pseudoexfoliation syndrome

2. **Visual field analysis/perimetry**
    - Visual field analysis is usually performed with Humphrey 30-2, threshold program
    - It consists of a bowl perimeter where stimuli of varying strengths and sizes are presented to the patient's visual field
    - Various patterns of the visual field are discussed previously
    - A Humphrey printout shows a greyscale (which shows a pictorial representation of the field analysis) and various values like mean deviation, standard deviation, pattern standard deviation, and glaucoma hemifield test
    - These values should be compared every time to note progression and response to treatment. Also, various progression analyses are available
3. **Optical coherence tomography**
    - Optical coherence tomography is used to quantify retinal fiber loss
    - It gives an image of various layers of the retina in a transverse section and thickness in each quadrant
    - Usually, in glaucoma, superior and inferior RNFL thinning in the peripapillary area is seen
    - Various grades of thinning are color-coded and hence it is easy to interpret. It is useful to pick up pre perimetric glaucoma (very early stages of ganglion cell loss which do not show on visual field analysis)
    - Nowadays macular thickness profile is coming of use in detecting very early changes, which shoes nasal macular thinning
4. **Pachymetry**
    - It is a measurement of corneal thickness. It can be done by ultrasound or AS-OCT
    - It is necessary to get a corrected IOP when IOP is measured using applanation
5. **Scanning LASER polarimetry and confocal laser scanning tomography**
    - Other methods used similar to optical coherence tomography
    - Helps to measure peripapillary retinal nerve fiber thickness

18. Explain the classification of angle-closure glaucoma according to traditional and the AIGS method. (3+2 marks)

Classification of Angle-closure Glaucoma

Older Classification

Latent phase	Defined as a primary angle-closure suspect Posterior trabecular meshwork is not visible on glonioscopy >270° of iridotrabecular contact
Prodromal phase	Characterized by periods of headache and colored halos lasting for a few minutes to hours, associated with stress and dim illumination It terminates by self due to miosis or sleep
Acute congestive glaucoma	Severe headache, vomiting, and prostration associated with a drop in visual acuity Corneal edema, angle closed in all quadrants, and raised IOP around 40–50 mm Hg The disc is edematous
Post-congestive glaucoma	Vogt's triad 1. Glaucomflecken (anterior subcapsular lenticular opacity) 2. Patches of iris atrophy 3. Slightly dilated non-reacting pupil (due to sphincter atrophy)
Chronic congestive glaucoma	All features are the same as POAG (quiet eye, optic nerve head changes, and visual field changes) but gonioscopy shows synechiae angle closure

Contd.

Absolute glaucoma	The eye is painful and completely blind (no light perception) Mild ciliary congestion Caput medusae: a few radial and enlarged vessels are seen in long-standing cases Decreased corneal sensations may be the only early sign The cornea becomes hazy, bullous keratopathy

AIGS Classification

Feature	Definition	Clinical Signs
Primary angle-closure suspect	Contact between peripheral iris and posterior trabecular meshwork	The posterior trabecular meshwork is not visible on gonioscopy >270° of iridotrabecular contact
Primary angle-closure	Peripheral iris obstructs trabecular meshwork causing raised intraocular pressure	Peripheral anterior synechiae Raised IOP Glaucomflecken Excessive pigments on angle structures
Primary angle-closure glaucoma	All above features + glaucomatous optic neuropathy + visual field changes	

19. Describe the management of angle-closure glaucoma according to its stage. (5 marks)

Management of Angle-closure Glaucoma

1. **Primary angle-closure suspect**
    - Periodic follow-up
    - Prophylactic LASER iridotomy
    - Long-term pilocarpine
    - LASER iridoplasty
    - Cataract extraction in presence of cataractous lens
2. **Primary angle-closure**
    - Similar to primary angle-closure suspect
    - Peripheral laser iridotomy. This is done to create a bypass for aqueous outflow when a pupillary block occurs
3. **Acute congestive glaucoma**

Immediate	• Make the patient lie down supine ▪ Intravenous or oral Acetazolamide 500 mg ▪ Topical steroid (Dexamethasone or Prednisolone) QID ▪ Topical timolol 0.5% ▪ Analgesics and antiemetics
After one hour	• Topical pilocarpine 2%, in both eyes, 4 applications every 15 minutes
After 30 more minutes	• Recheck IOP—if not fallen <35 mm Hg, oral or intravenous hyperosmotic drugs ▪ IV 20% mannitol 1g/kg over 45-60 min (1 drop per second) ▪ Oral 50% glycerol 1g/kg
Later	• Perform gonioscopy to understand the synechiae status of the eye • LPI in the affected eye, when IOP has dropped and is around 20–25 mm Hg • LPI in the fellow eye after communicating with the patient

4. **Chronic congestive glaucoma**
    - Similar to POAG
    - Trabeculectomy at areas where peripheral synechiae

20. Describe the clinical features of lens-induced glaucoma. (5 marks)

Type	Clinical features
Phacomorphic glaucoma	Severe pain, low vision Irregularly/regular shallow anterior chamber Iridodonesis and phacodonesis may be present Vitreous in the anterior chamber in subluxation cases
Phacolytic glaucoma	Severe eye pain Corneal edema, ciliary congestion, a deep anterior chamber with 4+ cells and severe flare, hypopyon, and a Morgagnian cataract
Phacoanaphylactic glaucoma	History of cataract surgery in the fellow eye, following trauma or surgery in the present eye Uveitis, high IOP
Lens particle glaucoma	Features of uveitis, hypopyon Lens matter is seen in the anterior chamber Raised IOP

21. Explain the mechanism by which uveitis causes glaucoma. (5 marks)

Mechanism by which Uveitis Causes Glaucoma

- Glaucoma associated with or following uveitis is very common
- There are many mechanisms of causing high IOP in uveitis
- It can be associated with an open-angle orclosed angle

Open-angle	Angle-closure
Changes in anterior chamber • Increased inflammatory cells, osmolality, red blood cells, and lens particles (in lens-induced glaucoma) causes decreased aqueous outflow Changes in angle • Trabeculitis (inflammation of cells in TM) is seen as a part of certain infectious uveitis (herpes infection) • Due to scarring of the trabeculum (post-inflammatory) • Steroid-induced glaucoma	• Due to secclusio pupillae-pupillary block glaucoma • Organized inflammatory debris in angle • Formation of peripheral anterior synechiae causes decreased aqueous outflow and glaucoma
Examples 1. Acute herpetic iridocyclitis 2. Fuch's heterochromic cyclitis 3. Posner–Schlossman syndrome 4. Lens protein uveitis 5. Juvenile idiopathic arthritis	Examples 1. Pupillary block glaucoma

22. Describe the etiology, clinical features, and management of malignant glaucoma. (2+1+2 marks)

Malignant Glaucoma

- Also known as a ciliary block or aqueous misdirection glaucoma
- In this condition, aqueous gets collected in the vitreous instead of the anterior chamber

Etiology

- Risk factors include hypermetropic eyes, nanophthalmos, and plateau iris configuration
- Following intraocular surgery, the tips of ciliary processes are anteriorly rotated and press against the equator of the lens in phakic eyes (cilio-lenticular block) or the intraocular lens (cilio-IOL block) or against the anterior hyaloid phase of vitreous in aphakic eyes (ciliovitreal block)
- This leads to a block of the forward flow of aqueous humor, which is diverted posteriorly and collects as aqueous pockets in the vitreous. As a result, the iris lens diaphragm is pushed anteriorly leading to secondary angle closure

Clinical Features

- This condition is characterized by a shallow anterior chamber with a raised intraocular pressure
- As against pupillary block glaucoma, the anterior chamber is diffusely flat
- Visual acuity is reduced due to corneal edema

Management

- A patent Nd-YaG iridotomy is required to diagnose malignant glaucoma
- Atropine is the first line of treatment as it pushes the iris lens diaphragm posteriorly by posterior rotation of the ciliary body
- Aqueous suppressants like beta-blockers, carbonic anhydrase inhibitors are used to decrease aqueous formation
- Nd YAG hyaloidotmy (creating an opening with LASER to release aqueous from the vitreous) is indicated in pseudophakic and aphakic eyes
- Chandler's procedure is a traditional procedure in which aqueous is aspirated from the vitreous using a needle and a syringe
- Vitrectomy is done if all the above measures fail

23. Elaborate peripheral iridotomy concerning indications, procedure, and complications. (5 marks)

Peripheral Iridotomy

An outpatient procedure in which an opening is created in the peripheral aspect of the iris using Nd: Yag laser

Indications

1. Treatment of an acute attack of angle-closure glaucoma
2. Post-inflammatory pupillary block glaucoma
3. To prevent an attack of angle-closure on the fellow eye
4. Prophylactically in latent angle closure
5. Malignant glaucoma sometimes may respond

Procedure

- The location of 11 o'clock or 1 o'clock on the peripheral iris is preferred
- The 12 o'clock position is avoided as gas bubbles released during the procedure may block the iridotomy
- Lower quadrants may lead to diplopia and glare
- The pupil is constricted using pilocarpine
- Some prefer to use apraclonidine before LASER

- An Abraham lens is used to fix the eye and focus the LASER
- A spot is chosen 1 mm from the periphery (too much in the periphery may lead to anterior synechiae formation) in between the crypts, LASER shot of 5–7 mJ strength is applied
- Successful iridotomy is marked by aqueous flowing out

Complications

1. Hyphema
2. Raised IOP
3. Corneal endothelial burn

SHORT ANSWERS

1. Differentiate between Megalocornea and Buphthalmos. (3 marks)

Feature	Megalocornea	Buphthalmos
Presentation	At birth	First year of life
Genetic	X linked recessive	Sporadic
Natural history	Non progressive	Progressive
Symptoms	None	Photophobia, lacrimation
Cornea	Enlarged, >13 mm Cornea globose (globe-shaped) Clear cornea	Can vary, asymmetric Flat cornea due to stretching Hazy due to edema
Anterior chamber	Deep	Deep
Axial length	Normal	High
Optic disc	Normal	Cupping or atrophy
Intraocular pressure	Normal	High

2. Describe the clinical features of primary congenital glaucoma. 3 marks)

- The presentation of childhood glaucomas is unique. Parents bring the child with excess tearing, child burying its head into pillow and blepharospasm. It is due to irritation of corneal nerves due to corneal edema
- The cornea appears hazy due to edema and is enlarged. It is called buphthalmos—bull eye. Normally, the diameter of the cornea is 10.5 mm; more than 13 mm is considered pathognomonic
- Haab's striae: Tears in Descemet's membrane are seen as parallel peripheral concentric tears
- The sclera is thinned due to increased pressure and appears blue
- The anterior chamber is deep
- Iris appears thinned and the lens is stretched
- The optic disc shows cupping or atrophy due to increased pressure
- Axial myopia is seen

3. Describe the clinical features of Juvenile glaucoma. (3 marks)

- Juvenile glaucoma is an autosomal dominant condition that presents in the age group of 10–35 years. It is due to a mutation occurring in the myocilin gene
- The risk factors for juvenile glaucoma include male sex, myopia, and African ancestry
- It is characterized by raised intraocular pressure in presence of open angles

- The patients are usually asymptomatic similar to POAG and discovered accidentally. Some may present with mild ocular pain and diminution of vision
- On examination, there is corneal edema, increased IOP (in the range of 40–45 mm Hg), and open angles on gonioscopy
- Optic disc changes and field changes usually are similar to POAG and a large subset of these patients go on to develop POAG in their adulthood

4. Explain the implications of (3 marks)

A. Water drinking test

B. Prone test

C. IOP phasing

A. Water-drinking Test

- It is a provocative test of historical interest
- A decrease in plasma osmolality due to increased water intake causes a decrease in aqueous outflow. As a result, IOP rises, more so in eyes with POAG
- After an overnight fast, baseline IOP is checked. Later, the patient is asked to drink 1L of water, following which IOP is noted every 15 minutes for 1 hour. IOP peaks at 15–30 minutes and returns to baseline level after 60 minutes
- A rise of 8 mm of Hg or more is said to be diagnostic of POAG

B. Prone Test

- After noting baseline IOP, the patient is made to lie in a prone position in a darkroom
- Dim illumination leads to mydriasis and the prone position helps to move the iris-lens diaphragm forward
- An increase in IOP of more than 8 mm Hg at the end of one hour is diagnostic of compromised angle

C. IOP Phasing

- It is the charting of regular IOP over 24 hours
- Charting of IOP every 2–4 hours for 24 hours is needed to know the diurnal variation of IOP. It is known as phasing
- Phasing is an important requirement for the diagnosis of POAG when the initial IOP may not be higher than 21 mm Hg
- Phasing helps to know the highest pressure, so the target pressure can be set accordingly

5. Define the target pressure. Describe the concept of neuroprotection. (1+2 marks)

Target Pressure

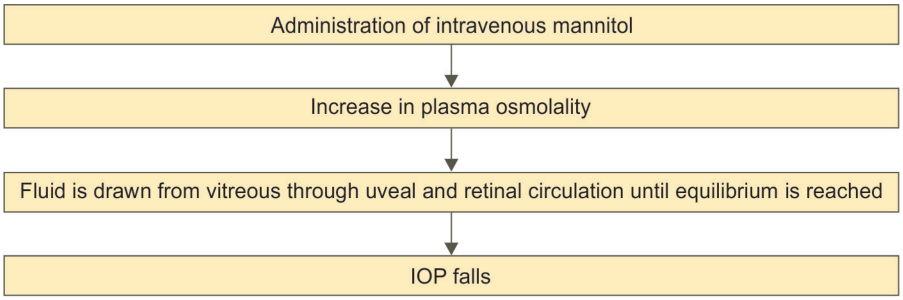
- Target pressure is an intraocular pressure at which progression of glaucoma is minimal
- It is based on factors like age of the patient, presence of risk factors, size of the cup, and family history of glaucoma

Neuroprotection

- It is a novel concept for treating glaucoma. It simply means protecting the remaining viable optic nerve head fibers
- It is an IOP-independent method of decreasing glaucoma progression

- Brimonidine is an alpha-agonist that has neuroprotective properties. It has been postulated to reduce the death of retinal ganglion cells from somatic, axonal, and dendritic degeneration. The dose is 0.2% BID and is indicated in low-tension glaucoma
- Another such drug is betaxolol. It is a selective beta 1 antagonist
- Mesenchymal stem cells are also found to be helpful in neuroprotection
- Various nerve growth factors like ciliary neurotrophic factor, brain-derived neurotrophic factor, and recombinant human nerve growth factor are being tried for neuroprotection in glaucoma
- Several drugs like ginkgo biloba, memantine, citicoline, calcium channel blockers, and nicotinamide are being tried for neuroprotection due to their effects on ocular blood flow

6. Explain the mechanism of action of mannitol. (3 marks)


Administration of intravenous mannitol
Increase in plasma osmolality
Fluid is drawn from vitreous through uveal and retinal circulation until equilibrium is reached
IOP falls

7. What are glaucoma drainage devices? Name two commonly used devices. (2 + 1 marks)

Glaucoma Drainage Devices

- Glaucoma shunts are synthetic devices that are placed in the subconjunctival space connecting to the anterior chamber
- They have a plate that goes into the subTenon/subconjunctival space and a tube that enters the anterior chamber.

Commonly Used Devices

1. Beervedlt valve
2. Ahmed valve

8. Give three indications for the use of glaucoma drainage devices. (3 marks)

1. Neovascular glaucoma
2. Glaucoma with aniridia
3. Intractable cases of primary and secondary glaucoma

9. Explain Bjerrum scotoma. (3 marks)

- Bjerrum's area is that part of the visual field that lies between 10–20° from fixation
- It is seen as an extension of Seidel's scotoma (Fig. 6.7.24)
- Early glaucomatous changes are seen as decreased sensitivity in this area and are referred to as Bjerrum's scotoma
- It is a relatively steep scotoma and is more commonly seen in the superonasal quadrant

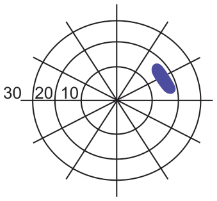


Fig. 6.7.24: Bjerrum's scotoma

10. Explain von Herick's grading of the angle of the anterior chamber. (3 marks)

- Depth is graded based on von Herick's grading. In this method, a narrow beam of light is focused on the peripheral cornea
- The distance between the beam reflecting off the cornea and the iris is considered as peripheral anterior chamber depth (PACD)

Grade 4 (Wide open angle)	PACD = ½ > CT
Grade 3 (Mild narrow angle)	PACD = ¼ to ½ CT
Grade 2 (Moderate narrow angle)	PACD = ¼ CT
Grade 1 (Extremely narrow angle)	PACD < ¼ CT
Grade 0 (Closed angle)	PACD = Nil

11. What is Fincham's test? Explain its use. (3 mark)

- Fincham test is a simple test using a stenopic slit and is performed in patients with colored halos
- It is a test with historical and academic interest
- In this test, a stenopic slit is placed in front of the eye with colored halos in different orientations (Fig. 6.7.25)
- If the halos break and reappear, then the colored halos of lenticular origin
- If they are constant, then it is corneal in origin as corneal edema is diffuse

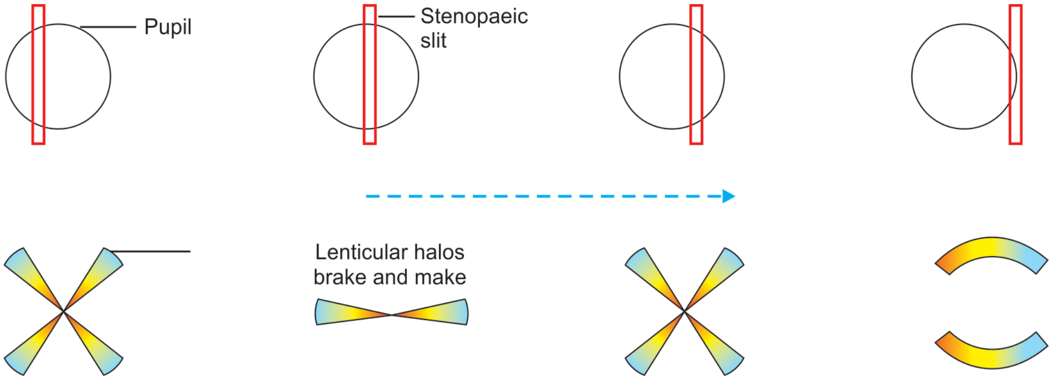


Fig. 6.7.25: Fincham test

12. Elucidate the features of Vogt's triad. (3 marks)

- Vogt's triad is seen as a consequence of acute congestive glaucoma and serves as a marker of a well-managed (IOP controlled) recent attack of angle closure
- It is characterized by:
  1. Glaucomflecken (anterior subcapsular lenticular opacity)
  2. Patches of iris atrophy
  3. Slightly dilated non-reacting pupil (due to sphincter atrophy)

13. Describe the anterior segment findings of pigmentary glaucoma. (3 marks)

Cornea	Krukenberg spindle: Pigments arranged in the form of a vertical spindle in the lower half of the cornea, on the endothelium
Anterior chamber	Deep
Iris	Mid peripheral transillumination defects Heterochromia in asymmetric cases
Angle	Wide-open angle, posterior insertion of the iris Sampaolesi's line: Deposition of pigments anterior to Schwalbe's line, more so in the inferior part.
Lens zonules	Eggert' line: pigments on the hyaloido-capsular ligament

14. Describe the clinical features of pseudoexfoliation glaucoma. (3 marks)

Symptoms

- The patients usually do not have any symptoms and are accidentally discovered
- A cataract is a common ocular association and thus may present with painless, progressive loss of vision

Signs

Cornea	Deposition of PEX and pigments on endothelium
Anterior chamber	Mild flare can be there
Iris and pupil	White powdery deposits on the pupillary border with a moth-eaten appearance at the sphincter The pupil does not dilate well
Lens	Due to the constant movement of the pupil, there is a preferential arrangement of this PEX material central material with mid-peripheral clearing and a peripheral ruff The higher tendency of nuclear cataract formation Zonules are weaker Cataract surgery is difficult in these cases- non dilating pupil, zonular weakness, and corneal decompensation
Gonioscopy	Pigment deposition may be confused with pigmentary glaucoma PEX material may be seen in angles

15. Explain the clinical features of various stages of neovascular glaucoma. (3 marks)

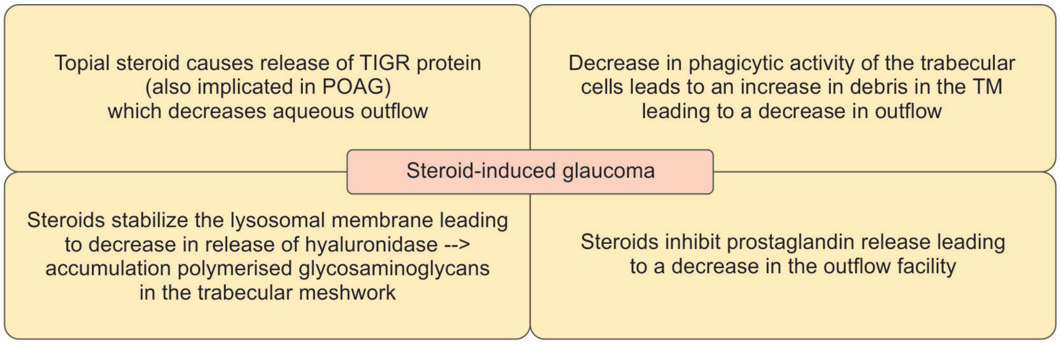
Preglaucomatous stage	It is an asymptomatic stage On examination, there is a presence of new vessels seen on the iris surface and in the angle. Fundus examination reveals the cause of neovascular glaucoma. (CRVO, PDR, etc)
Open-angle glaucoma stage	It is an asymptomatic condition characterized by raised intraocular pressure and the presence of new vessels in the iris and the angle There is progressive cupping
Secondary angle-closure stage	The patient presents with severe pain and diminished vision It is characterized by high IOP and corneal edema Gonioscopy reveals the presence of a neovascular membrane in the angle causing

16. Describe the principles of glaucoma shunt surgeries. (3 marks)

- Glaucoma shunts are synthetic devices that are placed in the subconjunctival space connecting to the anterior chamber
- They have a plate that goes into the sub-Tenon/subconjunctival space and a tube that enters the anterior chamber
- The aqueous is thus drained from the anterior chamber directly to the subconjunctival space and bypasses the natural course through the trabecular meshwork
- These are indicated when there are peripheral anterior synechiae that can interfere with a trabeculectomy. Also, they have better IOP control and are thus reserved for cases with intractable glaucoma
- Some types of glaucoma like ICE syndrome, neovascular glaucoma, and congenital glaucoma are well benefitted from glaucoma shunts
- Some shunts have valves (Ahmed glaucoma valve) which help in maintain IOP so that hypotony does not occur.
- They are indicated in cases of all types of glaucoma where trabeculectomy has failed

17. Explain the ways by which steroids can cause glaucoma. (3 marks)

The proposed mechanisms for steroid-induced glaucoma are:



Topial steroid causes release of TIGR protein (also implicated in POAG) which decreases aqueous outflow

Decrease in phagicytic activity of the trabecular cells leads to an increase in debris in the TM leading to a decrease in outflow

Steroid-induced glaucoma

Steroids stabilize the lysosomal membrane leading to decrease in release of hyaluronidase --> accumulation polymerised glycosaminoglycans in the trabecular meshwork

Steroids inhibit prostaglandin release leading to a decrease in the outflow facility

18. Define ocular hypertension. Enumerate four risk factors (according to the ocular hypertension treatment trial) that have been found to increase the progression to primary open-angle glaucoma. (1+2 marks)

Definition of Ocular Hypertension

- It is a subtype of POAG characterized by raised intraocular pressure (>21 mm Hg) with no visual field changes of optic nerve head changes
- Usually, the IOP is in the range of the late twenties

Risk Factors

Conversion to POAG has some risk factors such as (according to ocular hypertension treatment trial)

1. Positive water drinking provocative test
2. IOP constantly more than 28 mm of Hg
3. Central corneal thickness <555 µm
4. Myopia

COMPETENCY

OP6.8 ENUMERATE AND CHOOSE THE APPROPRIATE INVESTIGATION FOR PATIENTS WITH CONDITIONS AFFECTING THE UVEA

SHORT ESSAYS

1. Describe the investigations that are used in various types of uveitis. (5 marks)

A thorough history regarding the onset of the disease must be elicited by questioning the patient regarding past episodes, recurrences and drugs that are being used.

Anterior Uveitis

- Investigations are not required in first episode of unilateral iridocyclitis
- They are indicated in bilateral, recurrent or if associated with posterior uveitis
- The investigations should be individualized based on clinical presentation and not done as a battery of tests

Investigations

Blood

- Complete hemogram
- ESR; inflammatory pathology
- Blood sugars, uric acid
- RA factor, antinuclear antibodies, ASO (to rule out JIA associated uveitis)
- HLA typing in relevant cases

Others

- Urine routine and culture (to rule out Reiter's)
- Chest X ray for ruling out sarcoid, TB
- X-ray of spine to look for ankylosing spondylosis
- Mantoux, toxoplasmin and Kveim's test
- Colonoscopy in relevant cases to rule out Inflammatory bowel disease

Based on the type of presentation, the investigations necessary in a case of anterior uveitis are:

Nongranulomatous anterior uveitis in an adult	HLA B27 if recurrent iridocyclitis Chest X-ray, VDRL, FTA Abs
Granulomatous anterior uveitis in an adult	Chest X-ray, Mantoux test Serum angiotensin converting enzyme (ACE) levels, VDRL and FTA-ABS tests
Anterior uveitis in a child	ANA, HLA B 27
Suspected sarcoidosis in an adult or a child with granulomatous uveitis	Chest X-ray, HRCT chest Serum CA, ACE levels Whole body gallium scan, bronchoscopy with biopsy

Intermediate Uveitis

- Following a thorough ophthalmic examination, systemic associations of IU
- Multiple sclerosis: MRI brain, CSF analysis
- Lyme disease: Serology ELISA for borrelia antibody
- VDRL, FTA Abs: To rule out syphilis
- Sarcoidosis: Serum calcium, serum angiotensin converting enzyme, chest X-ray
- To rule out tuberculosis: Chest X-ray and Mantoux test

Posterior Uveitis

Infective

- It is individualized based on the clinical appearance of the lesion
- For toxoplasmosis: serum IgG and IgM *Toxoplasma*
- For tuberculous uveitis: Mantoux test, X-ray chest
- For syphilitic uveitis: VDRL, RPR, treponema immobilization test, MRI brain
- For CMV retinitis: Aqueous and vitreous IgM
- PCR of aqueous

Noninfective

- Ocular investigations: FFA, B scan, ICG angiography
- HLA typing to rule out autoimmune vasculitis
- Sarcoidosis: Serum Ca, ACE levels, whole body Ga scan
- For disseminated choroiditis: MRI brain
- For periphlebitis: ANA, serum calcium and ACE (for sarcoid)

2. Enumerate the indications for investigations in uveitis. Describe the investigations for anterior granulomatous uveitis. (2+3 marks)

Indications for Investigations in Uveitis

- All cases of uveitis need to undergo a complete ophthalmic examination
- The first episode of anterior uveitis does not need to be investigated, especially if the fundus is normal
- Indications for investigations are:
    1. Recurrent iridocyclitis
    2. Bilateral iridocyclitis
    3. Any iridocyclitis in a child
    4. Any posterior uveitis
    5. Any intermediate uveitis
    6. Pan-uveitis need to be investigated

Investigations for Anterior Granulomatous Uveitis

Acute Granulomatous Uveitis in an Adult

- To rule out TB
    - Chest X-ray, Mantoux test
- To rule out sarcoidosis
    - Serum angiotensin-converting enzyme (ACE) levels
    - HRCT chest and whole-body Ga scan
- To rule out syphilis
    - VDRL and FTA-ABS tests

Acute Granulomatous Uveitis in a child

- To rule out JIA associated uveitis
    - HLA B27
    - ANA
- To rule out sarcoidosis
    - Chest X-ray, serum ACE levels

SHORT ANSWERS

1. Enlist the investigations of ocular sarcoidosis. (3 marks)

Supportive Diagnosis

- Cutaneous anergy
- Raised serum calcium, angiotensin-converting enzyme
- Whole-body gallium scan
- BAL (bronchoalveolar lavage) fluid for CD4: CD8

Confirmatory Diagnosis

Histopathology of a lymph node suggestive of noncaseating granuloma

2. Enumerate investigations for intermediate uveitis. (3 marks)

Following a thorough ophthalmic examination, systemic associations of IU as mentioned before have to be ruled out

1. Multiple sclerosis: MRI brain, CSF analysis
2. Syphilis: VDRL, FTA-ABs
3. Lyme disease: Serology ELISA for borrelia antibody
4. Sarcoidosis: Serum calcium, serum angiotensin-converting enzyme, chest X-ray

3. Enumerate the investigations that may help in diagnosing the cause for a solitary retinitis lesion. (3 marks)

Causes for a solitary retinitis patch include ocular toxoplasmosis, toxocariasis, syphilis, and tuberculosis

Toxoplasmosis	• Serum IgG and IgM toxoplasmosis • Aqueous IgG and IgM toxoplasmosis • HIV and CD4+ counts
Toxocariasis	• IgM toxocara
Syphilis	• Serum VDRL, FTA-Abs • Demonstration of live treponemes in aqueous from paracentesis
Tuberculosis	• Mantoux test, chest X-ray • Aqueous or vitreous tap positive for *Mycobacterium tuberculosis* by PCR • Histopathology of hilar or cervical lymph nodes • Sputum smear • Quantiferon

COMPETENCY

OP6.9 CHOOSE THE CORRECT LOCAL AND SYSTEMIC THERAPY FOR CONDITIONS OF THE ANTERIOR CHAMBER AND ENUMERATE THEIR INDICATIONS, ADVERSE EVENTS AND INTERACTIONS

SHORT ESSAYS

1. Describe the treatment of anterior uveitis. (5 marks)

Anterior uveitis or iridocyclitis is treated with:

A. Nonspecific therapy
B. Specific therapy

A. Nonspecific Therapy

1. **Cycloplegics**
   - These are the mainstay of treatment in anterior uveitis
   - Atropine is the best drug available
   - It is available as 1% atropine sulfate drops and ointment
   - It acts in the following ways
     - Relaxes the ciliary body and decreases pain
     - Dilates the pupil and thus decreases the contact between lens and pupil
     - Prevents the formation of synechiae and breaks the already formed ones
     - Reduces inflammation by decreasing vascularity and permeability
     - Increases absorption of other drugs
   - Other drugs that are used are 2% homatropine, 1% cyclopentolate, tropicamide-phenylephrine (mild cases)
   - Alternatively, subconjunctival injection of adrenaline, atropine, and procaine (mydricain) can be given in severe cases
   - They must be given for at least 2–3 weeks even after the inflammation subsides to prevent recurrences
2. **Corticosteroids**
   - The steroids that can be used may range from potent steroids like prednisolone and Dexamethasone to milder forms like loteprednol, fluorometholone, and medrysone
   - Depending on the severity, the steroids are instilled frequently and then tapered gradually
   - A few maintain a small dose of steroid in recurrent cases
   - It may also be given as subconjunctival or sub-Tenon injection in severe cases
   - Systemic steroids are only indicated in a few cases of bilateral iridocyclitis or resistance to topical steroids
3. **Other supportive drugs**
   - If associated with complications, their treatment is required as well

Cystoid macular edema	Topical NSAIDs
Complicated cataract	Cataract extraction
Glaucoma	Antiglaucoma medications like carbonic anhydrase inhibitors and beta-blockers can be used In cases of pupillary block, not resolving with mydriasis, peripheral iridotomy should be performed

B. Specific Therapy

- If caused by an infectious agent, then antimicrobial is used. For example, ATT for tuberculosis, acyclovir in herpes zoster uveitis, penicillin in syphilis, and so on
- NSAIDs (Sulfasalazine) are used in uveitis associated with ankylosing spondylosis

2. Describe the management of intermediate uveitis. (5 marks)

Intermediate Uveitis

Investigations

- Following a thorough ophthalmic examination, systemic associations of IU as mentioned before have to be ruled out
- Multiple sclerosis: MRI brain, CSF analysis

- Lyme's disease: Serology ELISA for *Borrelia* antibody
- Sarcoidosis: Serum calcium, serum angiotensin-converting enzyme, chest X-ray

Management

- 80% of cases resolve spontaneously, hence may not require treatment
- In severe non-resolving conditions, steroids are given in the form of subtenon injections or oral administration
- Side effects of steroids include steroid-induced glaucoma and cataract
- If associated with multiple sclerosis, interferon-beta may be of help
- In presence of neovascularization in the peripheral retina, laser photocoagulation is indicated
- Vitrectomy: Indicated when
  - There is no response to steroids
  - Vitreous opacities significantly decreasing vision
  - Unresolved cystoid macular edema
  - Vitreous hemorrhage
  - Tractional retinal detachment
  - Epiretinal membrane
- Accompanying endo-LASER can be done if there is associated neovascularization

3. Describe the treatment of noninfectious posterior uveitis. (5 marks)

Treatment of Noninfectious Posterior Uveitis

Noninfectious posterior uveitis is treated with corticosteroids and immunomodulators.

Corticosteroids

- Steroids can be given in the form of periocular steroids and systemic steroids
- Periocular steroids can be given as subtenon or orbital floor injections
- Systemic steroids are given in the inflammatory dose (1 mg/kg of prednisolone)
- Along with periocular steroids
  - Sarcoidosis: Systemic steroids
  - Behcet's disease: Systemic steroids, chlorambucil
  - Ankylosing spondylosis: Systemic indomethacin and Aspirin
  - VKH and sympathetic ophthalmia: Systemic steroids
  - When unresponsive to steroids, immunomodulators are to be used

Immunosuppressants

- Many immunosuppressants are useful in controlling inflammation in uveitis, especially if associated with a systemic component
- The immunosuppressives are of many types
- Antimetabolites (methotrexate, mycophenolate mofetil, azathioprine)
- T-cell inhibitors (tacrolimus and cyclosporine)
- Alkylating agents (cyclophosphamide and chlorambucil)

Biologicals

- Mainly reserved for chronic relapsing and nonresponsive, noninfectious uveitis
  1. **Daclizumab (IL 2 antagonist)**
     - It is a CD20 monoclonal antibody
     - Interleukin 2 is an important part of the lymphokine receptor system

    - Blocking its action is brought about by daclizumab
    - It is given as 1–2 mg/kg subcutaneously or intravenously every 2–4 weeks
  2. **Infliximab (TNF alpha antagonist)**
    - It is approved for use in rheumatoid arthritis, ankylosing spondylitis, and Crohn's disease
    - It is used in uveitis refractory to conventional therapy and is useful in Behcet's syndrome, juvenile idiopathic arthritis, inflammatory bowel disease, VKH
    - It is given as an intravenous injection of 3–5 mg/kg dose, weekly
- Others are adalimumab (TNF alpha antagonist), etanercept (TNF alpha antagonist), rituximab (CD20 inhibitor), and anakinra (interleukin 1 receptor antagonist)

4. Describe the role of steroids in uveitis. Explain its side effects. (2+3 marks)

Uveitis

Role of Steroids

- Steroids act in uveitis by decreasing inflammation
- Steroids enter the anterior chamber through the conjunctival vessels and diffuse through the cornea
- Commonly used steroids are dexamethasone, prednisolone, loteprednol, fluorometholone, and medrysone
- Depending on the severity, the steroids are instilled frequently and then tapered gradually
- A few maintain small doses of steroids in recurrent cases. It may also be given as subconjunctival or subtenon injection in severe cases
- Systemic steroids are indicated in a few cases of bilateral iridocyclitis or resistance to topical steroids

Side Effects

Topical Steroids

- Steroid-induced glaucoma
- Steroid-induced cataract
- Increased propensity to fungal keratitis

Systemic

- Steroid-induced cataract
- Steroid-induced glaucoma
- Systemic hypertension and hyperglycemia
- Secondary Cushing's syndrome
- Osteoporosis
- Increased weight gain
- Easy bruising

5. Describe the procedure of evisceration. (5 marks)

Procedure of Evisceration

- Removing the contents of the eyeball is called evisceration
- It is indicated in panophthalmitis, bleeding anterior staphyloma, and expulsive choroidal hemorrhage

Simple Evisceration

- Conjunctival peritomy and tenonectomy are done circumferentially
- The eyeball is entered with a stab wound at the limbus
- The wound is extended on both sides to remove the cornea
- Intraocular contents are scooped out with an evisceration scoop
- Care must be taken to remove all choroid
- The remaining sclera is sutured in purse-string fashion with an implant or otherwise. The conjunctiva is closed over it
- A prosthesis is placed at a later stage

Frill Evisceration

- Conjunctival peritomy is done circumferentially
- The eyeball is entered with a stab wound at the limbus
- The wound is extended on both sides to remove the cornea
- Intraocular contents are scooped out with an evisceration scoop
- Care must be taken to remove all choroid
- The extraocular muscles are severed
- The remaining sclera is excised using scissors leaving a frill of 3 mm around the optic nerve
- Conjunctiva and Tenon's fascia are closed

6. Explain the methods of delivering periocular steroids. (5 marks)

Methods of Delivering Periocular Steroids

1. **Posterior sub-Tenon**
   - After instilling topical proparacaine, a cotton pledget soaked in paracaine is placed in the superior fornix
   - 40 mg/ml of triamcinolone is loaded in a 2 ml syringe and a 25 guage needle is inserted
   - The patient is advised to look inferonasally steadily
   - The needle is inserted in the superotemporal quadrant of the globe in the bulbar conjunctiva and the needle is advanced in a zig-zag manner along the contour of the globe
   - After withdrawing to check for vascular entry, the drug is delivered
2. **Inferior transseptal injection (orbital floor injection)**
   - The skin of the lower lid and maxillary area is cleaned with spirit
   - 40 mg/ml of triamcinolone is loaded in a 2 ml syringe and a 25 guage needle is inserted
   - The patient is advised to maintain a straight gaze
   - Through the skin of the lower lid, at the junction of the outer third and inner two-thirds, the needle is inserted along the orbital floor. The orbital septum is pierced and the needle is moved in a slight zig-zag manner in order to avoid perforation of the sclera
   - After withdrawing to check for vascular entry, the drug is delivered

SHORT ANSWERS

1. Outline the treatment of idiopathic anterior uveitis. (3 marks)

Anterior uveitis or iridocyclitis is treated with topical steroids and cycloplegics.

Topical Cycloplegics

- These are the mainstay of treatment in anterior uveitis.

- Atropine is the best drug available. It is available as 1% atropine sulfate drops and ointment
- Other drugs that are used are 2% homatropine, 1% cyclopentolate, tropicamide-phenylephrine (mild cases)
- Alternatively, subconjunctival injection of adrenaline, atropine, and procaine (mydricain) can be given in severe cases
- They must be given for at least 2–3 weeks even after the inflammation subsides to prevent recurrences

Corticosteroids

- The steroids that can be used may range from potent steroids like prednisolone and Dexamethasone to milder forms like loteprednol, fluorometholone, and medrysone
- Depending on the severity, the steroids are instilled frequently and then tapered gradually
- A few maintain a small dose of steroid in recurrent cases. It may also be given as subconjunctival or subtenon injection in severe cases
- Systemic steroids are only indicated in a few cases of bilateral iridocyclitis or resistance to topical steroids

2. Name three biologicals used in uveitis. (3 marks)

1. Daclizumab (IL-2 antagonist)
2. Infliximab (TNF alpha antagonist)
3. Adalimumab (TNF alpha antagonist)

3. Describe the treatment of ocular toxoplasmosis. (3 marks)

- Treatment is not mandated in all cases of ocular toxoplasmosis
- In the adult form, the infection usually arises from the proximity of a healed lesion
- Indications for therapy in toxoplasmosis
    1. Lesion close to the macula
    2. Lesion close to the papillomacular bundle
    3. Lesion close to the optic nerve
    4. Lesion close to a major arterial arcade
    5. Significant vitritis
    6. Immunocompromised individuals
- The treatment is continued till the resolution of the retinitis patch is seen
- Systemic corticosteroids are started under the cover of antimicrobials

Drug	Dose	Contraindications
Clindamycin	300 mg QID	Hepatotoxicity Renal toxicity Pseudomembranous colitis
Cotrimoxazole (not as efficacious as classical therapy)	800 mg/160 mg	G6PD deficiency Sulfa allergy Bone marrow suppression
Azithromycin	500 mg/day	GI disturbances Useful in pregnancy
Spiramycin	1.5 million IU	GI hypersensitivity Safest in pregnancy
Atovaquone	750 mg QID	Caution in liver failure

4. Enumerate three indications of vitrectomy. (3 marks)

1. Retinal detachment
2. Endophthalmitis
3. Vitreous hemorrhage

COMPETENCY

OP6.10 COUNSEL PATIENTS WITH CONDITIONS OF THE IRIS AND ANTERIOR CHAMBER ABOUT THEIR DIAGNOSIS, THERAPY AND PROGNOSIS IN AN EMPATHETIC MANNER IN A SIMULATED ENVIRONMENT

SHORT ANSWER

1. Justify the need for timely diagnosis and treatment of JIA-associated uveitis. (3 marks)

- JIA is associated with chronic, unilateral/bilateral, anterior uveitis
- It is also known as Still's disease and can present in the eye with complications of uveitis like cataracts, band-shaped keratopathy, and even phthisis bulbi
- The inflammation tends to be very mild and chronic and the children may not report the symptoms like pain, photophobia, blurred vision, and redness, which is usually seen in other causes of uveitis. This phenomenon is also called 'white-eye uveitis'
- On a careful slit-lamp examination, one can find evidence of uveitis in the form of cells, flare, and even posterior synechiae
- Children of the female gender, with ANA positivity, oligoarthritis, and age of onset of arthritis below 4 years have a higher risk of developing arthritis

Screening

- Meticulous screening with a slit-lamp examination and IOP measurement is required as these patients are also prone to hypertensive uveitis
- Diagnosis is based on the presence of cells and flare

Management

- They are treated with topical steroids and cycloplegics
- Atropine is best avoided in these children for the fear of developing amblyopia and posterior synechiae as the pupil remains dilated. Short-acting cycloplegic-mydriatics (cyclopentolate, tropicamide) are preferred
- Methotrexate is the gold-standard systemic treatment for JIA-associated anterior uveitis

CHAPTER

7

Lens

COMPETENCY

OP7.1 DESCRIBE THE SURGICAL ANATOMY AND THE METABOLISM OF THE LENS

SHORT ESSAYS

1. Describe the surgical anatomy of the lens. (5 marks)

- The lens is a biconvex structure placed behind the iris and in front of the vitreous
- It is present a fossa in the anterior face of the vitreous called the 'patellar fossa'
- Its diameter is around 9–10 mm and its thickness varies from 4–5 mm and is held circumferentially by zonules, which arise from the ciliary processes
- It has an anterior surface, a posterior surface, and an equator (Fig. 7.1.1)
- The posterior surface is more curved

Parts of the Lens

1. **Capsule**
    - It is a thin hyaline membrane that is prominent anteriorly and at the equator
    - It is very thin at the posterior pole with a mere 6–10 μm thickness
    - It is the thickest in the pre-equatorial region
    - Surgical implication
        - The anterior capsule is removed during cataract surgery in a continuous curvilinear manner to make a saucer-shaped capsule for placement of the IOL
        - The posterior capsule is retained in the surgery
        - In case of rupture of the posterior capsule, vitreous prolapses into the anterior chamber and placement of IOL becomes difficult

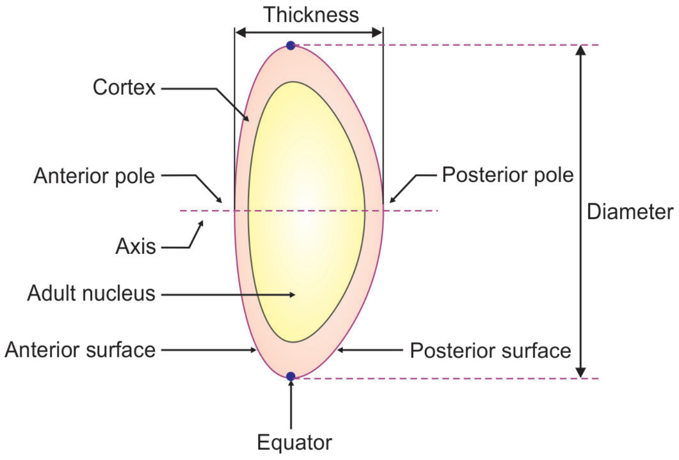


Fig. 7.1.1: Anatomy of the lens

2. **Cortex**
    - The cortex is formed by young lens fibers which are nothing but elongated epithelial cells
    - The fibers are arranged in a fashion so that they meet at the poles
    - Anteriorly, their line of intersection forms an erect 'Y' whereas, posteriorly they form an inverted 'Y'
    - These are called suture lines
    - Surgical implication
        - It gets hydrated during surgery and is removed by aspiration
        - If cortex is not fully removed, it contributes to inflammation
3. **Nucleus**
    - It is the central core of the lens and is formed by densely packed lens fibers that have lost their nuclei
    - The oldest of the fibers, embryonic is the deepest
    - It is followed by fetal, infantile, and adult nuclei and represents the time at which they were laid
    - Surgical Implication
        - Nucleus hardens with age, and diameter increases
        - In nuclear cataract, it is better to have a large capsulotomy to deliver the lens in SICS
        - In phacoemulsification, nuclear cataracts need a higher energy to break
4. **Suspensory ligaments of the lens (zonules of Zinn)**
    - These fibers are attached to the equator and arise from the ciliary body
    - These hold the lens in a stretched position and enable the ciliary muscle to act on it
    - Surgical implication
        - Zonular strength decreases with age and certain pathologies like pseudoexfoliation syndrome
        - Zonular loss causes irregular capsular bag and thus makes IOL placement difficult

2. A taxi driver looks at his watch while driving. Explain the changes that take place in the lens during this process. (5 marks)

- Looking at his watch during driving needs accommodation of the lens
- Accommodation is an active process in which changes are occurring in the lens to bring divergent rays arising from a proximal object focus onto the retina
- During this process, the total power of the eye increases
- The changes that take place during accommodation are in the following order (Fig. 7.1.2)

1. **Slackening of zonules**
    - According to the most accepted theory of Helmholtz, there is the contraction of the ciliary muscles that cause relaxation of the ciliary zonules
    - The zonules are attached to the edges of the lens capsule
    - Normally, the zonules are tensed and hold the capsule taut. During accommodation, they become slack or lax
2. **Changes in the capsule**
    - Due to the laxity of the zonules, there is a laxity in the anterior capsule of the lens
3. **Changes in the lens**
    - There is an increase in the anterior curvature of the lens
    - The radius of curvature decreases from 11 to 6 mm
    - The lens becomes more convex

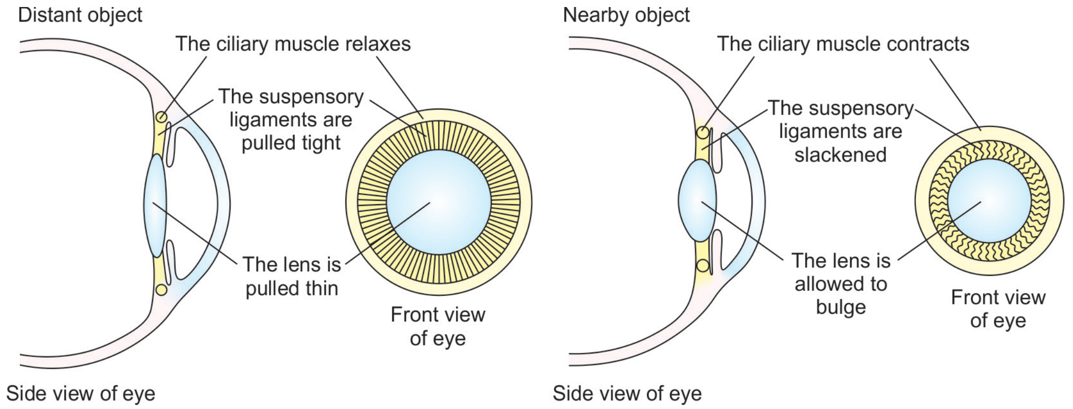

Distant object
The ciliary muscle relaxes
The suspensory ligaments are pulled tight
The lens is pulled thin
Front view of eye
Side view of eye
Nearby object
The ciliary muscle contracts
The suspensory ligaments are slackened
The lens is allowed to bulge
Front view of eye
Side view of eye

Fig. 7.1.2: Changes during accommodation

- There is an increase in the anteroposterior diameter of the lens
- There is an anterior movement of the lens, and so, the depth of the anterior chamber decreases
- The equatorial diameter of the lens decreases

3. Explain the biochemical composition of the lens and the ways by which energy is generated. (2+3 marks)

Biochemical Composition of the Lens

- The lens is made of 65% water and 34% proteins, and 1% of other substances like amino acids, lipids, and carbohydrates
- The protein content of the lens is densest than any other part of the body
- It has two main types of proteins

1. **Albuminoids**
   - Water insoluble
2. **Crystallins**
   - Water-soluble
   - It is made of alpha, beta, and gamma crystallins
   - Beta crystallins are found in abundance

Energy Generation in the Crystalline Lens

- Lens requires a continuous supply of energy (ATP) as
  - It is actively growing and requires constant cell division at the pre-equatorial region
  - Active transport of ions and amino acids and to maintain lens dehydration
  - Continuous protein and glutathione synthesis.
- Since the crystalline lens is avascular, it is dependent on its metabolism on chemical exchanges with the aqueous humor
- There is an active Na-K-ATPase that drives out Na ions for the exchange of K ions
- As a result, Na concentration is low inside the lens. Due to this, there is relative dehydration maintained inside the lens.
- Glutathione is synthesized within the lens which has antioxidant properties
- The glucose that enters the cell is mainly used for maintaining the biochemical activity of the lens

- The epithelium is a highly metabolic part of the lens whereas the nucleus is relatively inert

80%	Glycolysis
3%	Krebs cycle
14%	HMP shunt pathway
3%	Sorbitol pathway

- 80% of generated ATP–used up by epithelium
- 10–20% of generated ATP–used for protein synthesis

4. Explain the factors that maintain the transparency of the lens. (5 marks)

Factors Maintaining the Transparency of the Lens

1. The lens is an avascular structure. There are no blood vessels in the lens. As a result, there is a uniform passage of light in the lens
2. A single layer of the epithelium in the anterior surface of the lens does not interfere with the passage of light.
3. There are no nerves within the lens
4. Lack of nuclei and organelles in the lens fibrils
5. Nature of arrangement of lens fibrils
    - The distance between the two fibrils is smaller than the wavelength of light. So, there is no light dispersion
    - The fibers are arranged in a fashion so that they meet at the poles. Anteriorly, their line of intersection forms an erect 'Y' whereas, posteriorly they form an inverted 'Y'. These are called suture lines. This is an optically advantageous property of the lens fibers
    - The lens fibrils are not more than 10 nm in diameter, hence they do not interfere with the passage of light
6. The semipermeable character of the lens capsule
7. Pump mechanism of lens fiber membranes: maintain relative dehydration by pumping out water
8. A high concentration of glutathione and antioxidants remove oxidative material

5. Describe the embryology of the lens with a neat diagrammatic representation. (5 marks)

Embryology of the Lens (Fig. 7.1.3 to 7.1.8)

- The development of the eye begins on the 22nd day of gestation when the embryo has 8 pairs of somites and is 2 mm in length
- The specialized part of the neural plate becomes thickened (lens placode) on either side and then the intervening tissue deepens to form a sulcus called the optic sulcus

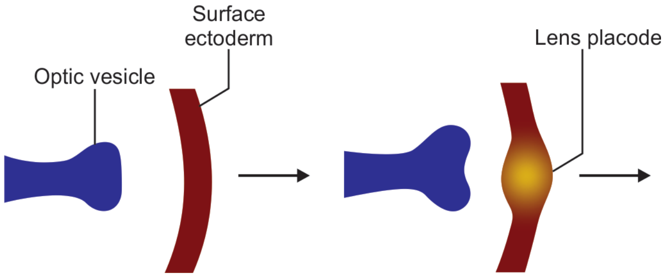


Fig. 7.1.3: Formation of lens placode

- The lateral sides of the sulcus form bulbous projections and approach the overlying surface ectoderm, these are called the optic vesicle. The overlying surface ectoderm forms a lens pit (Fig. 7.1.4)
- The proximal part narrows and becomes a tubular chord- optic stalk
- The surface ectoderm overlying the optic vesicle becomes thickened and invaginated the optic vesicle and thus leads to an optic cup (2 layers) formation (Fig. 7.1.5)
- Within the cup, a portion of the surface ectoderm is pinched off and forms the lens placode
- It gets detached from the surface ectoderm and forms a lens vesicle (around 6 weeks)
- The lens forms by differential growth of cells of the anterior and posterior layer of the lens vesicle
- Primary lens fibers are formed by the elongation of cells of the posterior wall of the lens vesicle, thus obliterating the lens cavity. These form the embryonic nucleus and are formed up to the third month of gestation (Fig. 7.1.6)
- Secondary lens fibers are formed by the proliferation of pre-equatorial cells of the anterior epithelium which remain active throughout life. (Fig. 7.1.7)
- These fibers are laid down concentrically (Fig. 7.1.8) to the embryonic nucleus and are named as

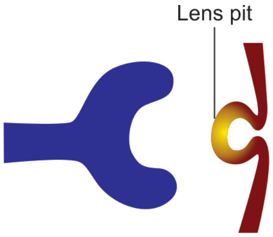
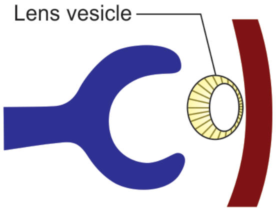
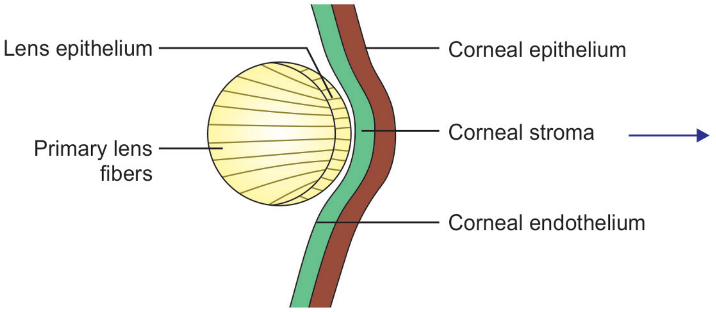


Fig. 7.1.4: Formation of lens pit


Lens vesicle

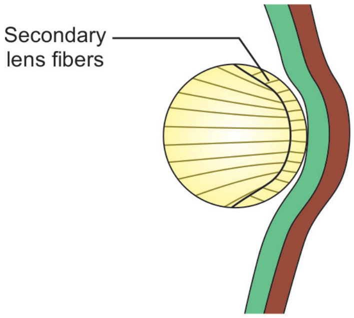
33 days gestation

Fig. 7.1.5: Formation of lens vesicle


Lens epithelium
Primary lens fibers
Corneal epithelium
Corneal stroma
Corneal endothelium

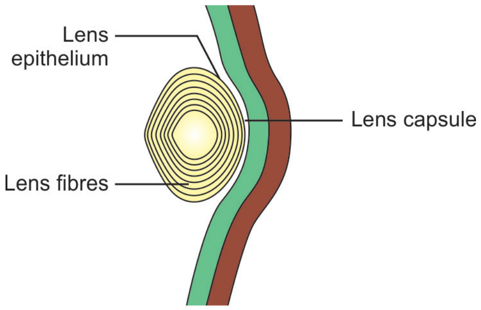
50 says gestation

Fig. 7.1.6: Formation of primary lens fibers


Secondary lens fibers

52 says gestation

Fig. 7.1.7: Formation of secondary lens fibers


Lens epithelium
Lens capsule
Lens fibres

56 days gestation

Fig. 7.1.8: Concentric arrangement of fibers in a mature lens

- Fetal nucleus: 3rd to 8th month
- Infantile nucleus: 40th week of gestation to puberty
- Adult nucleus: After puberty
- Cortex: Superficial lens fibers of adult lens

6. The human eye can be compared to a camera. Describe the role of the human crystalline lens in the eye as an optical system. (5 marks)

Role of the Human Crystalline Lens in an Optical System

1. **Contribution to the passage of light**
   - The lens occupies the central aspect of the globe or the eyeball
   - Its transparency allows the passage of light rays that have refracted through the cornea and the aqueous humor
   - As a result, the light rays are allowed to fall on the retina, which is the light-sensitive layer of the eyeball
   - When there is opacification of the lens, it leads to a cataract and there is a decrease or distortion of the light that travels in the eye
2. **Contribution to the total power of the eye**
   - The human crystalline lens behaves as a convex lens with approximately 15–20D (one-fourth of the total power of the eyeball). This is second to the cornea
   - The lens helps to converge the rays entering it onto the retinal surface
   - The power of the lens is determined by its thickness (increases with age), the refractive index of the fibers (also increases with age), the anterior surface curvature (dynamic), and its position. Physiological variation in the curvature and position happens during accommodation
   - Examples of pathological variants in the above-mentioned points are:
     1. Nuclear sclerosis—induces myopia
     2. Cortical sclerosis—induces hypermetropia
     3. Lenticonus—increased curvature of the anterior lens surface causes myopia
     4. Anterior subluxation of lens causes myopia
3. **Role in accommodation**
   - The human crystalline lens plays a central role in accommodation.
   - Accommodation is defined as a dynamic process by which changes occur within the optical system of the eye to bring divergent rays arising from a proximal object of interest are brought to focus onto a point on the retina
   - The accommodative power decreases with age
   - It is around 14–16D at birth and decreases to about 1–2D at the age of 50 years. This results in presbyopia
   - Thus, presbyopia is a physiological decrease in the amplitude of accommodation
4. **Absorption of UV light**
   - The visible spectrum of electromagnetic radiation is from 350 to 700 nm in wavelength.
   - The cornea absorbs those radiations lesser than 300 nm
   - The lens absorbs UV rays with a wavelength lesser than 350 nm
   - This is proposed to be protective of the macula from ARMD

SHORT ANSWERS

1. Describe the arrangement and functions of zonular fibers. (2+1 marks)

Arrangement of Zonular Fibers

- Suspensory ligaments of the lens (zonules of Zinn)
  - These fibers are attached to the equator and arise from the ciliary body
  - These fibers are arranged in three groups
    1. Posteriorly arising fibers arising from pars plana get inserted anterior to the equator
    2. Anteriorly arising fibers are inserted posteriorly to be inserted posterior to the equator
    3. Centrally arising fibers pass from the summits of the ciliary processes attach at the equator
  - Traditionally, the zonular fibers have been classified as (Fig. 7.1.9)
    1. Orbiculoposterior capsular fibers
    2. Orbiculoanterior capsular fibers
    3. Cilioposterior capsular fibers
    4. Cilioequatorial capsular fibers
- The newer concept, however, describes the parts of zonules as (Fig. 7.1.10)
  1. Pars orbicularis
  2. Zonular plexus
  3. Zonular fork
  4. Anterior, equatorial, and posterior zonular limb

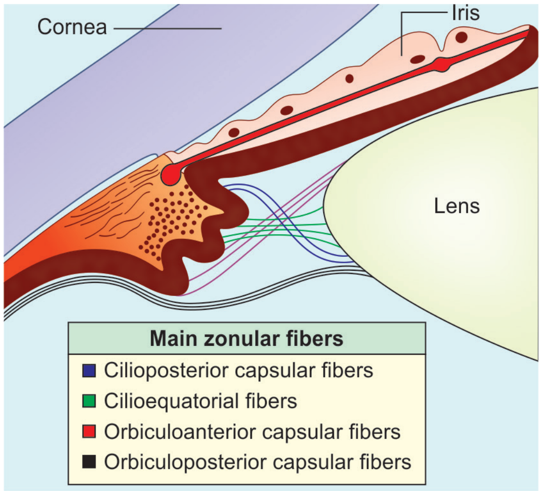

Cornea
Iris
Lens
Main zonular fibers
🟦 Ciliposterior capsular fibers
🟩 Cilioequatorial fibers
🟥 Orbiculoanterior capsular fibers
⬛ Orbiculoposterior capsular fibers

Fig. 7.1.9: Older classification of the zonular fibers

Functions of Zonular Fibers

1. These hold the lens in a stretched position and keep the refractory status of the lens regular
2. They enable the ciliary muscle to act on it. Therefore, they take part in accommodation

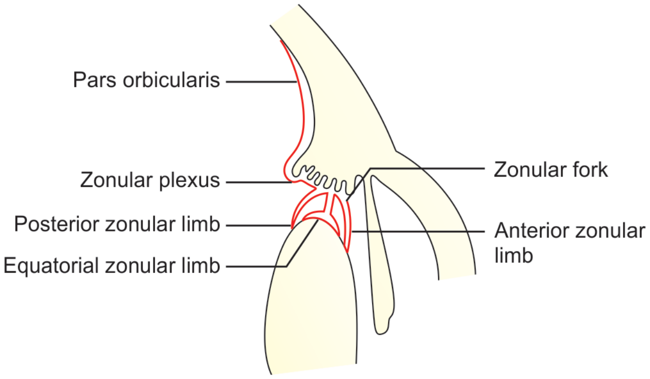

Pars orbicularis
Zonular plexus
Posterior zonular limb
Equatorial zonular limb
Zonular fork
Anterior zonular limb

Fig. 7.1.10: Newer classification of the zonular fibers

2. Draw a neat, labeled diagram of the cross-section of the human crystalline lens with its parts. (3 marks)

See Figure 7.1.11

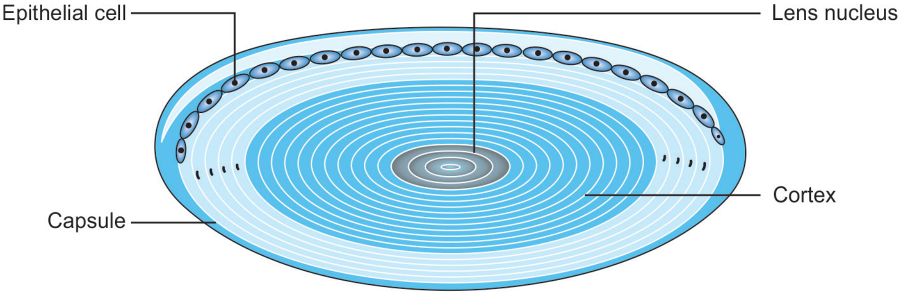


Fig. 7.1.11: Structure of human crystalline lens

3. Explain the importance of antioxidant mechanisms in the lens. (3 marks)

- The lens has several protective antioxidant mechanisms to protect itself from the various oxidative products generated during routine metabolism
- The oxidative molecules are superoxide anions (O_2^-), hydrogen peroxide, and peroxides
- The following mechanisms contribute to maintaining an antioxidant environment in the lens
  - Glutathione reductase is an enzyme that reduces sulfhydryl glutathione
  - Superoxide dismutase enzyme catalyzes
  - A high level of ascorbic acid acts as a scavenger of superoxide ions and converts them into lesser oxidative hydrogen peroxide
- With increasing age and exposure to the UV rays from the sun
  - There is a fall in these antioxidant mechanisms
  - There is a decrease in the synthesis of glutathione. Also, the lens becomes less permeable to reduced glutathione
  - Also, there is a decrease in the levels of glutathione reductase that causes decreased levels of glutathione
- Due to a failure of these protective mechanisms, there is a tendency to formation of cataract, and it is termed senile cataract

COMPETENCY

OP7.2 DESCRIBE AND DISCUSS THE AETIOPATHOGENESIS, STAGES OF MATURATION, AND COMPLICATIONS OF CATARACT

LONG ESSAY

1. A two-month-old child is brought to the ophthalmologist with bilateral whitish opacity in the pupil.

A. Define leukocoria. (1 mark)

B. Enumerate five differential diagnosis for the same in a child. (2 marks)

C. Explain how would you assess this child's eyes? (3 marks)

D. Describe the investigations that you would like to perform. (3 marks)

E. Name one chromosomal abnormality that is associated with a congenital cataract. (1 mark)

A. Definition of Leukocoria

A whitish discoloration seen within the pupil.

B. Differential Diagnosis for the Same

1. Congenital cataract
2. Retinoblastoma
3. Toxocariasis
4. Persistent fetal vasculature
5. Coats' disease

C. Assessment of a Child with Bilateral Leukocoria

1. **History taking**
   - It begins with a good history taking from the parents.
   - Whether the opacity has been present at the time of birth or developed over a course of time
   - History of any illness in the mother during the antenatal period, use of any teratogenic drugs has to be elaborated
   - History regarding the child's behavior concerning vision, as assessed by the parents or guardians should be asked. Some parents may notice a delay in the development of milestones
   - History of other systemic illnesses and drugs (esp. corticosteroids) must be elaborated
2. **General examination and systemic examination**
   - Should be performed to rule out associated features
   - Example
     - Features like antimongoloid slant, brachycephaly point towards a diagnosis of Down syndrome
     - Tetany can be present in hypocalcemia and there can be associated developmental cataract.
     - Presence of sensorineural hearing loss and heart defects along with leukocoria points towards congenital rubella syndrome
3. **Ocular examination**
   - Involves assessment of ocular posture, visual acuity, anterior segment evaluation, and fundus examination
   - The presence of squint and other anomalies should be assessed
   - Visual acuity assessment can be a task when the child is too young to cooperate or has other neurological abnormalities.
   - Anterior segment evaluation should be done using a slit lamp. If not possible in a very young child, a surgical loupe can be used
   - Fundus examination is mandatorily performed after complete mydriasis
   - This also helps in understanding the morphology of the cataract. If the fundus is not visible, a B-scan should be performed

D. Investigations in a case of Bilateral Leukocoria

- Laboratory investigations are needed for bilateral, nonhereditary cataract
- Blood investigations
  - Blood glucose, calcium, and phosphorus
  - Red blood cell transferase and galactokinase levels

- TORCH infections: ELISA for IgM
  - Urine assay for reducing substances after feeding
  - Screening for amino acids in urine (Lowe syndrome)

E. Chromosomal Abnormality

- Trisomy 21: Down syndrome

SHORT ESSAYS

1. A 75-year-old man presents with a gradual painless diminution of vision in one eye. On examination, he is found to have an immature cataract. He is a known hypertensive and a diabetic for 25 years.

A. Identify the risk factors for cataracts in this patient. (1 mark)

B. Explain the stages of cortical cataracts and their clinical features. (4 marks)

A. Risk Factors for Cataracts in this Patient

1. Age
2. Diabetes

B. Stages and Clinical Features of Cortical Cataract

A senile cortical cataract passes along these stages of maturation

1. **Stage of lamellar separation**
   - Earliest change of cataract formation and is reversible
   - There is an appearance of a demarcation line between the fibers and is visible only on the slit lamp
   - There are no visual symptoms

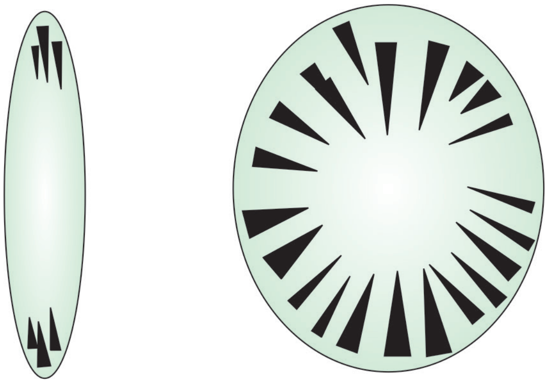


Fig. 7.2.1: Cuneiform cataract

2. **Stage of incipient cataract**
   - It is of two types—cuneiform and cupuliform
   - In cuneiform cataracts, wedge-like opacities are seen starting from the equator towards the center (Fig. 7.2.1)
   - They usually begin in the inferonasal quadrant
   - These opacities do not interfere with vision
   - A cupuliform cataract is characterized by a saucer-shaped opacity in the visual axis and posterior subcapsular area
   - This opacity is visually significant as it lies very close to the nodal point

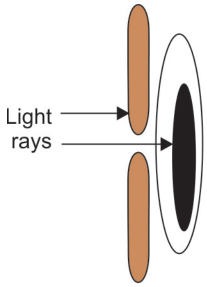


Fig. 7.2.2: Concept of iris shadow

3. **Stage of immature cataract**
   - It is the opacification of a part of the lens with the surrounding clear lens
   - The lens is greyish white
   - As a result, there is mild to moderate interference with visual acuity
   - Due to a part of the clear lens between the opacity and the iris, the iris casts a shadow on the lens. This phenomenon is called iris shadowing (Fig. 7.2.2)

4. **Stage of mature cataract**
    - The whole lens is opacified and the lens is now pearly white
    - Since there is no clear part of the lens, the iris does not cast a shadow, so the iris shadow is absent
    - Visual acuity is very poor, as poor as hand movements
5. **Stage of hypermature cataract**
    - This stage occurs after further cataractous changes occur to a mature cataract
    - It can occur in two morphological forms
        1. *Morgagnian cataract*
            - It is an intumescent (swollen) type of hypermature cataract, where the cortex is liquefied and the brown nucleus sinks at the bottom
            - The anterior chamber is shallow
            - The capsule is tense and may develop minute pores through which lens matter may leak to induce phacolytic glaucoma
        2. *Sclerotic hypermature cataract*
            - The cortex gets disintegrated and the lens is shrunken due to leakage of water
            - The anterior capsule gets wrinkled and thickened
            - Due to shrinkage of the lens, the anterior chamber becomes deep, and the iris becomes tremulous (iridodonesis)

2. Individuals residing at high altitudes have been found to have a higher incidence of cataracts. Explain the interplay of various genetic and environmental factors in the formation of senile cataracts. (5 marks)

- The most common cause of cataract is senility
- The risk factors affecting the onset and maturation of cataract are

Genetic Factors

1. **Heredity:** It plays an important role in the incidence, age of onset of cataract, and maturation of cataract
2. **Myopia:** Myopia has been implicated in several ocular conditions; with cataracts and glaucoma being the most common
3. **Diabetes:** Increases the risk of cataracts and is the most important cause of presenile cataract

Environmental Factors

1. Exposure to sunlight
    - The UV rays in the sunlight are postulated to cause a cataract
    - The range of UV rays (290–320 nm) causes oxidative damage to the lens fibers causing a cataract
    - The downregulation of the NA-K-ATPase causes swelling of the lens fibers due to the imbibition of fluid
    - This theory has been proven in various experiments and also evident clinically as a higher incidence of cataracts in people living at higher altitudes like the Himalayas (Fig. 7.2.3)

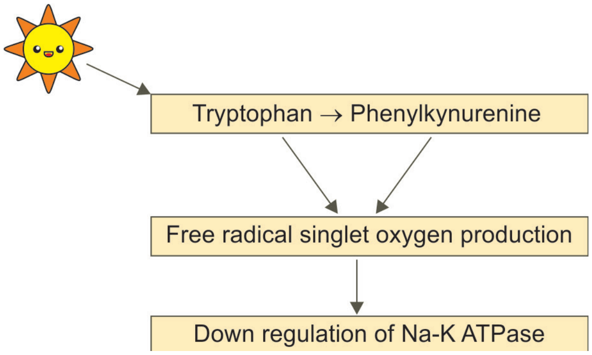

Tryptophan → Phenylkynurenine
Free radical singlet oxygen production
Down regulation of Na-K ATPase

Fig. 7.2.3: Pathogenesis of cataracts formation due to sunlight

2. Smoking is an important risk factor for the early development and maturation of cataracts
3. Diet: An imbalance of dietary contents has been found to have an increased incidence of cataracts. Poor socioeconomic status adds to malnutrition and these individuals have a higher incidence of cataracts
4. Severe dehydration: Dehydration leads to malnutrition, acidosis, high blood urea, all of which contribute to an osmotic imbalance between ions and aqueous

3. Explain various morphological types of cataracts with the help of a neat and labeled diagram. (5 marks)

Morphological Types of Cataracts (Fig. 7.2.4)

1. **Polar:** It is the opacification of the pole only. It is of two types: Anterior polar and posterior polar
2. **Capsular:** Opacification of capsule alone
3. **Subcapsular:** Immediately inner to the capsule—anterior and posterior subcapsular. A posterior subcapsular cataract is the most common type of senile cataract
4. **Pyramidal:** A projecting polar cataract with an underlying subcapsular cataract
5. **Cortical cataract:** Opacification of the cortex
6. **Supranuclear:** Cataract of deep parts of the cortex
7. **Nuclear cataract:** Opacification of the nucleus

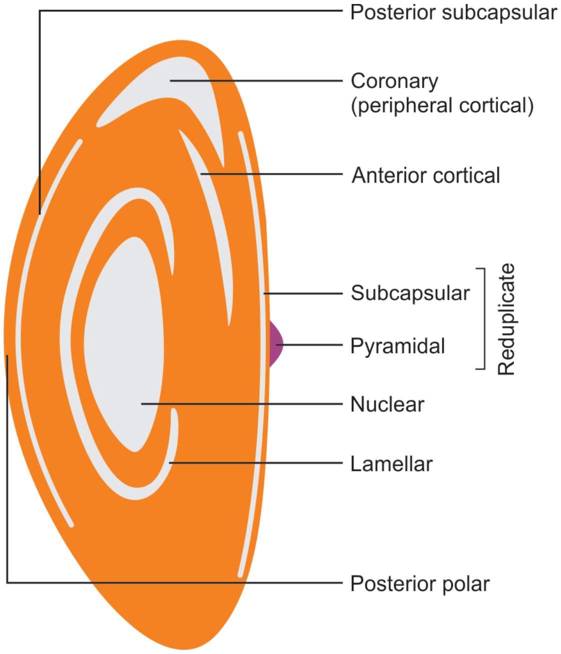


Fig. 7.2.4: Morphological classification of cataract

4. Explain the various manifestation of congenital cataracts. (5 marks)

Congenital cataract presents in the following ways:

1. **Leukocoria**
   - White reflex in the pupil. It is the most common presentation
   - Leukocoria has many differential diagnoses and is very important to differentiate these, as some of these are life-threatening

Retinoblastoma	Organized vitreous hemorrhage
PHPV (persistent primary hyperplastic vitreous)	Astrocytic hamartoma
Toxocariasis	Endophthalmitis
Retinopathy of prematurity	Norrie's disease
Coats' disease (exudative retinal detachment)	Medullated nerve fibers
Medulloepithelioma	Congenital cytomegalovirus infection

2. **Esotropia/exotropia**
   - Due to decreased vision in one eye, there may resultant convergent squint (usually less than 2–3 years of age) or exotropia (in children >4 years of age)
3. **Failure to develop certain developmental milestones**
   - May be observed by the parents like lack of social smile, delay in fine motor skills, or fine skills. Some may notice an absence of a social smile especially when the cataract is bilateral and visually significant
4. **Accidental discovery**
   - Some institutions and countries employ a routine red-reflex test or Bruckner's test at regular intervals such as the day of immunization. It may reveal absence or decreased glow on distant direct ophthalmoscopy by the examiner. Some parents may discover an absence of red reflex in a photograph
5. **Asymptomatic**
   - Many anterior or small (<3 mm) cataracts may remain asymptomatic without causing any visual disturbances

5. Explain how pediatric cataract differs from adult cataract in terms of presentation and management. (5 marks)

Adult Cataract and Pediatric Cataract

Adult cataract	Pediatric cataract
Clinical presentation	
• Most common presentation is diminution of vision	• As children may not be able to communicate the visual diminution, it is difficult to pick up cases of pediatric cataracts • They may present with leukocoria, strabismus, failure to develop milestones, or amblyopia
• Vision assessment is the most important part of evaluation	• Vision assessment is very difficult in infants, thus have to resort to other methods like preferential looking tests, optokinetic nystagmus, and VEP
• No risk of amblyopia or strabismus due to cataract • Cataract extraction is not emergency	• Amblyopia and strabismus: incidence of amblyopia increases with a delay in surgical removal of the lens due to stimulation deprivation. Hence, a unilateral cataract should be treated on an emergency basis
Management	
• Calculation of IOL is based on keratometry, axial length, anterior chamber depth, and Lens constant	• Calculation of IOL power is tricky in the pediatric age group, as there is a myopic shift during the growth of the eye and more so following intraocular surgery • The IOL power which is calculated is under-corrected by 10% in children of 2–8 years of age and under-corrected by 20% in children less than 2 years of age to overcome the myopic shift
• Can be performed in local anesthesia	• There is always a need for general anesthesia
• Low incidence of postoperative glaucoma	• Higher incidence of postoperative complications
• Follow-up is for 6 weeks	• Longer follow-up is needed in pediatric cataracts • Treatment of amblyopia is required
• Sutures are not required	• The eyeball is more resilient, so sutures have to put

Contd.

Adult cataract	Pediatric cataract
• Chances of posterior capsular opacification decreases with age	• There are higher chances of posterior capsular opacification • Hence, after cortex removal, a part of the posterior capsule is removed along with anterior vitrectomy • If IOL is being placed in the same sitting, it is fixed into the posterior capsulotomy
• The lens is emoved by phacoemulsification (nucleus needs to carved and broken)	• The lens is very soft and is easily removed by irrigation and aspiration • Another method is pars plana lens aspiration using a vitrector • A scleral incision is made 4 mm behind the limbus and the lens is aspirated using a vitrector. Anterior vitrectomy is done. The rim of the capsular bag is left in place for IOL placement

6. Contrast cortical and nuclear cataracts concerning the pathogenesis, clinical features, and surgical consideration. (5 marks)

Feature	Nuclear cataract	Cortical cataract
Pathogenesis	• There is a decrease in antioxidant mechanisms that occur within the lens. There is also an increase in the insoluble component of the lens proteins (albuminoids) and compaction of the lens fibers • There is associated melanin deposition derived from the amino acids in the lens	• With age, there is a decrease in the function of the active transport pump mechanism of the lens that leads to the reversal of the Na^+-K^+ ratio within the lens. As a result, the lens fibers imbibe more fluid • Hydration of the lens fibers causes denaturation of the lens proteins and opacification of cortical lens fibers
Clinical features	• Presents after the 7th decade • Causes index myopia • Associated with changes in the color of the lens • There is no stage of hypermaturity or liquefaction	• Presents in 5th to 6th decade • Causes induced hypermetropia • No change in color of the lens • There is a stage of hypermaturity associated with the liquefaction of the cortex and the sinking of the nucleus to the bottom of the lens
Surgical consideration	• The lens becomes increasingly harder and larger • Increased chances of capsular tear during lens delivery • Higher power is required during phacoemulsification	• The lens becomes soft • Increased chances of run-away of the capsulorhexis due to high pressure of the fluid • Phacoemulsification is easier and requires less power

SHORT ANSWERS

1. Describe the complications of cataracts. (3 marks)

The two important complications of cataracts are phacomorphic and phacolytic glaucoma.

Phacomorphic Glaucoma

- Due to increased size of the lens (intumescent cataract) or due to subluxation of the lens
- The lens pushes the iris forward and blocks the angle leading to secondary angle-closure glaucoma
- Presents with severe pain, low vision, irregularly/regular shallow anterior chamber; Iridodonesis and phacodonesis may be present; Vitreous in the anterior chamber in subluxation cases
- It is treated by removing the cataract after decreasing the intraocular pressure

Phacolytic Glaucoma

- It is due to rupture of the lens capsule in a hypermature morgagnian cataract leading to a release of lens proteins into aqueous
- Lens proteins are high molecular weight and are phagocytosed by macrophages which can occlude trabecular meshwork to lead to secondary open-angle glaucoma
- Presents with Severe eye pain, corneal edema, ciliary congestion, a deep anterior chamber with 4+ cells and severe flare, hypopyon, and a Morgagnian cataract
- It is treated with topical steroids, cycloplegics, and antiglaucoma medications. Following this, it is treated by cataract surgery

2. Explain the biochemical basis for cataractogenesis in (1 marks each)

A. Nuclear cataract
B. Cortical cataract
C. Sugar cataract

A. Nuclear Cataract

- In nuclear cataract, there is an intensification of age-related changes that occur in the lens
- There is a decrease in anti-oxidant mechanisms that occur within the lens.
- There is also an increase in the insoluble component of the lens proteins (albuminoids) and compaction of the lens fibers
- There is associated melanin deposition derived from the amino acids in the lens

B. Cortical Cataract

- With age, there is a decrease in the function of an active transport pump mechanism of the lens that leads to the reversal of the Na+-K+ ratio within the lens. As a result, the lens fibers imbibe more fluid
- Hydration of the lens fibers causes denaturation of the lens proteins and opacification of cortical lens fibers
- Also, there is a decrease in the level of amino acids due to which there is reduced synthesis of proteins in lens fibers

C. Sugar Cataract

Sugar cataract refers to cataracts due to galactosemia and type I diabetes

Galactosemic cataract	Type I diabetes
• Due to deficiency of Galactose 1-phosphate uridyl transferase and galactokinase • As a result, there is an accumulation of galactose, a monosaccharide • The lens fibers imbibe more fluid • Hydration of the lens fibers causes denaturation of the lens proteins and opacification of cortical lens fibers.	• When an excess of glucose accumulates, it is reduced to sorbitol by the enzyme aldose reductase • As a result, there is an increase in the osmolarity of the lens fibers causing imbibition of fluid and hydration of lens fibers • This causes denaturation of lens fibers and opacification

3. Define cataractogenesis. Classify cataracts based on etiology.

(1+2=3 marks)

Definition of Cataractogenesis

The process by which a transparent crystalline lens develops opacities leading to obstruction to the passage of light leading to a noticeable visual deficit.

Classification of Cataract

- Congenital and developmental cataract
- Acquired cataract
  1. Senile cataract—most common cause
  2. Traumatic cataract
  3. Complicated cataract
  4. Metabolic cataract
  5. Electric cataract
  6. Radiational cataract
  7. Drug-induced: Steroid-induced, miotic induced, copper and siderotic cataract
  8. Dermatogenic or syndermatotic cataract (associated with skin diseases)
  9. Associated with osseous diseases

4. A 33-year-old woman presents with a unilateral mature cataract. Enumerate three probable causes for presenile cataracts. (3 marks)

1. Type 1 diabetes mellitus
2. Uveitis
3. Trauma

5. Enumerate three drugs that can cause cataracts. (3 marks)

1. Corticosteroids
2. Miotics like echothiophate
3. Amiodarone

6. Explain the clinical features of a zonular cataract. (3 marks)

- A zonular cataract is the most common form of pediatric cataract. It is also named a lamellar cataract as it affects only one particular lamella of the nucleus
- It is inherited autosomal dominant, may be associated with vitamin D deficiency or rubella in the late gestational period
- It affects a particular 'zone' of the lens with central and peripheral clear areas. It has spokes-like riders in the periphery (Fig. 7.2.5)
- Almost always bilateral

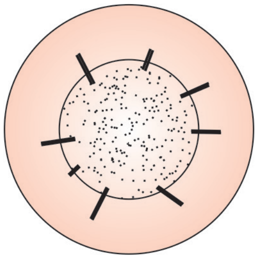

Zonular cataract
(stearing wheal appearance)

Fig. 7.2.5: Zonular cataract

7. Name the stages of cortical cataract. (3 marks)

1. Stage of lamellar separation
2. Stage of incipient cataract
3. Stage of immature cataract
4. Stage of mature cataract
5. Stage of hypermature cataract

8. Explain the clinical features of traumatic cataracts. (3 marks)

- A cataract can occur following blunt trauma or penetrating trauma
- In blunt trauma, due to the transmission of compression forces, the posterior pole of the lens is affected
- Opacification begins here and has a floral pattern. Hence, the name rosette cataract
- It may progress to total cataract

- In penetrating injury, a breach in the anterior capsule leads to imbibition of aqueous into the lens fibers leading to swelling of the lens and opacification. Also, there may associated subluxation

9. Enumerate three causes of complicated cataracts. 3 marks)

1. Uveitis
2. Retinitis pigmentosa
3. Long-standing retinal detachment

10. Explain the grades of nuclear cataracts. (3 marks)

Grades of Nuclear Cataract

- According to Lens opacification classification II, nuclear cataract is graded into 5 stages based on color

Grade	Color	Consistency during surgery
1	Grayish white	Soft
2	Yellowish	Mild hard
3	Amber	Hard
4	Brown (cataracta brunescens)	Very hard
5	Black (cataracta nigra)	Ultra-hard

- A nuclear cataract is graded based on the color and opalescence according to LOCS III into 6 grades and is based on comparison to standard photographs

COMPETENCY

OP7.3 DEMONSTRATE THE CORRECT TECHNIQUE OF OCULAR EXAMINATION IN A PATIENT WITH CATARACT

SHORT ESSAYS

1. Describe the evaluation of cataracts using the slit lamp. (5 marks)

- A patient with a cataract needs a thorough ophthalmic evaluation in the form of visual acuity testing, slit-lamp examination, tonometry, fundus examination, and B-scan, if necessary
- A good slit-lamp examination of both eyes is necessary to understand the type of cataract and to find associated abnormalities

Assessment of the Anterior Segment

Cornea

- Look for the health of all layers of the cornea
- Evidence of dry eye, old opacities, dystrophies, endothelial health are important for the outcome of cataract surgery
- Anterior chamber
- The presence of even a mild amount of inflammation as evidenced by cells and flare should be used as a pointer to uveitis and surgery should be deferred
- Depth of the anterior chamber is also important. If the anterior chamber is uneven, then subluxation of the lens and the presence of anterior synechiae should be looked for

Iris

The presence of iris atrophic patches, nevi, new vessels, pigmentation is necessary to rule out associated pathologies.

Pupil

- Normal pupillary light reflex is necessary in cases of nonvisualization of the fundus
- Also, the surgeon must know the amount of pupillary dilatation before surgery
- Dilatation of the pupil well before the surgery helps to ascertain the amount of dilatation and presence of posterior synechiae if any

IOP measurement

It is important to rule out glaucoma.

Gonioscopy

It is only performed in selective cases when there is an indication like the presence of new vessels on the iris or newly discovered glaucoma.

Assessment of the Cataract

1. **Polar:** Anterior polar and posterior polar
2. **Capsular:** Opacification of capsule alone
3. **Subcapsular:** Immediately inner to the capsule—anterior and posterior subcapsular
4. **Pyramidal:** A projecting polar cataract with an underlying subcapsular cataract
5. **Cortical cataract:** Opacification of the cortex
6. **Supranuclear:** Cataract of deep parts of the cortex
7. **Nuclear cataract:** Opacification of the nucleus

- Cortical, nuclear, and subcapsular cataracts are the most commonly encountered types of senile cataracts
- Grading of the cataract is done based on LOCS III classification
  - Lens opacification classification system is a method of grading cataracts based on slit-lamp examination and retroillumination
  - It is useful only in senile cataracts and is based on a set of images of cortical, nuclear, and subcapsular cataracts.
  - A nuclear cataract is graded based on color and opalescence into 6 categories
  - Cortical cataracts and posterior subcapsular cataracts are categorized into 5 grades based on retroillumination images
  - This is a standardized grading and thus helps avoid confusion
- Types of cortical cataract are differentiated based on the following tests

Feature	Immature	Mature	Hypermature
Distant direct ophthalmoscopy	Red glow present	Red glow absent	Red glow absent
Iris shadow	Present	Absent	Absent
Color of lens	Grayish white	Pearly white	Milky white

- Other features
  - A posterior polar cataract may be associated with a posterior capsular defect
  - Phacodonesis indicates zonular weakness

2. Describe the macular function tests and their utility in cataracts. (5 marks)

Macular tests are performed in cases where the fundus is not visualized and if the visual loss is more than the amount of cataract.

In Case of Very Poor Vision and Nonvisualization of Fundus

- Perception of light: It is a test for optic nerve functioning
- Projection of rays:
  - It is a test for the integrity of the retina outside the posterior pole
  - It is performed by shining a thin beam of light from various quadrants and asking the patient to identify the direction of the light
  - If PR is inaccurate, there is a chance of the presence of retinal detachment
- Pupillary reaction: Swinging flashlight to detect RAPD
- Entoptic visualization: A pen torch is placed on the closed lid and the patient perceives retinal vasculature as branches. If there is any pathology in the retina, black spots may be seen by the patient
- LASER interferometry
- VEP
- ERG- foveal flicker sensitivity

In Case of Moderate Loss of Vision with Nonvisualization of Macula Only

- Photostress recovery test
  - In this test, after testing visual acuity, a bright source of light is shown for 15 seconds
  - 45–60 seconds postexposure, the visual acuity returns to normal
- Two-point discrimination
  - In this test, opaque cardboard with two holes 2 inches apart is placed 2 feet from the eye to be examined.
  - A light is shone from behind the cardboard.
  - If the patient perceives it as two images, then his macula is normal
- Potential acuity meter
  - In this, the pupil is fully dilated, and the Snellen chart is projected through a small clear area of the immature cataract with a +12D placed in front of the cataractous eye
  - The acuity is judged based on the number of lines recognized by the patient
- Maddox rod
  - It consists of horizontal cylinders.
  - When asked to view a point source of light, it is visible as a straight vertical line

SHORT ANSWERS

1. Explain the potential acuity meter. (3 marks)

- A potential acuity meter is a method of assessing the potential visual outcome after eventless cataract surgery when the fundus cannot be visualized
- in this test, the pupil is fully dilated, and the Snellen chart is projected through a small clear area of the immature cataract through the slit lamp with a +12D placed in front of the cataractous eye
- This test is useful in eyes with nuclear cataracts

2. Explain the concept of iris shadow. (3 marks)

- The iris shadow is demonstrated as a crescentic shadow seen on the peripheral aspect of the lens on the side from which light is shone
- In an immature cataract, there is opacification of a part of the lens with a surrounding clear lens
- Due to a part of a clear lens between the opacity and the iris, the iris casts a shadow on the lens (Fig. 7.3.1)
- When light rays are thrown on the pupil, the iris casts a shadow of itself on the opaque part of the lens
- The iris shadow is absent in a mature cataract

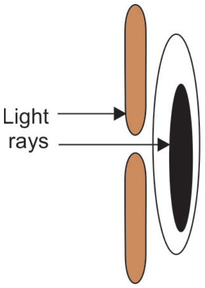
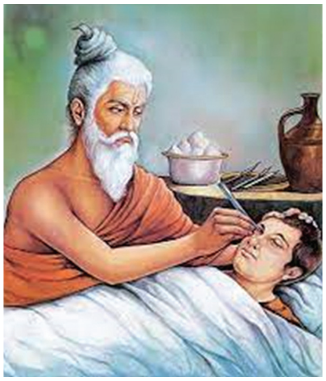


Fig. 7.3.1: Iris shadow

COMPETENCY

OP7.4 ENUMERATE THE TYPES OF CATARACT SURGERY AND DESCRIBE THE STEPS, INTRAOPERATIVE AND POSTOPERATIVE COMPLICATIONS OF EXTRACAPSULAR CATARACT EXTRACTION SURGERY



The author would like to begin this section by paying a tribute to the Ancient Indian Father of Surgery—Sushruta, 6000 BC. The Sushrutha Samhita has 18 chapters devoted to the eye and was the first to perform cataract surgery by couching. With no slit-lamp, autoclave, and microscope, couching was the only option for cataracts. It involves introducing an instrument to push the lens into the vitreous. The visual acuity improves slightly for the patient to ambulate and see his food.

To know more

https://www.aao.org/senior-ophthalmologists/scope/article/sushruta

LONG ESSAYS

1. Cataract surgery is the most commonly performed procedure worldwide.

A. Explain the steps of small incision cataract surgery. (3 marks)

B. Compare and contrast small incision cataract surgery and phacoemulsification (3 marks)

C. Describe four postoperative complications of cataract surgery. (4 marks)

A. Steps of Small Incision Cataract Surgery

- This technique is the most practiced in developing nations and has good visual recovery compared to ECCE
- The steps are:
  - After painting and draping the eye, a wire speculum is placed
  - Superior rectus bridle suture is applied.
  - A superior fornix-based conjunctival flap is raised and episcleral vessels are cauterized
  - A 6–7 mm long straight or frown incision is made 2 mm posterior to the limbus.
  - The incision is converted into a tunnel without entering the anterior chamber

- A sharp blade-keratome is used to enter the anterior chamber in a zig-zag manner such that the wound is self-sealing
- Trypan blue dye is injected to stain the anterior capsule and capsulotomy is performed in one of the three methods as mentioned above
- Hydrodissection, nucleus delivery, and cortical irrigation are similar to ECCE
- Before nucleus delivery, the tunnel should be converted to a funnel to deliver out the nucleus
- PCIOL is placed into the bag and the anterior chamber is maintained
- There are no sutures needed as the wound is self-sealing. Sutures may be needed if the iris is floppy or there was a premature entry into the anterior chamber
- The speculum is removed and the eye is closed and padded

B. Small Incision Cataract Surgery (SICS) vs. Phacoemulsification

Feature	SICS	Phacoemulsification
Size of incision	6–7 mm	3–4 mm
Type of anesthesia	Peribulbar/retrobulbar	Topical is sufficient
Postoperative recovery	24–48 hours	12–24 hours
Learning curve	Easy	Difficult
Apsulotomy	Can opener or curvilinear	Has to be continuouscurvilinear capsulorhexis
Nucleus removal	Delivered out using a vectis	Chopped and aspirated
Postoperative astigmatism	More	Less
Procedure cost	Low	High
Instrument availability	Easy	Difficult

C. Complications of Cataract Surgery

1. Hyphema

- Collection of blood in the anterior chamber. Visual acuity depends on the severity of hyphema
- This can happen due to a bleed from the scleral wound or due to inadvertent iridodialysis
- It usually subsides and gets absorbed. Treatment is supported with cycloplegics and topical corticosteroids
- If it is associated with raised intraocular pressure, antiglaucoma medications are given

2. Striate keratopathy

- It is the most common finding in the postoperative period and appears as lines of varying dimensions in the deep cornea. They are folds along the Descemet's membrane that appear due to sudden decompression of the anterior chamber
- They may be dense if there was frequent shallowing of the anterior chamber intraoperatively
- These are nothing but folds in the Descemet's membrane due to changes in the pressure of the eyeball
- They disappear with time and need no specific treatment

3. **Corneal edema**
    - They may be associated with dense striate keratopathy and occur because of endothelial damage or increased intraocular pressure
    - Incidence of edema is higher in older individuals, those with a larger nucleus, lower endothelial count, and intraoperative complications
    - Hyperosmotic solutions are given to decrease corneal edema
4. **Iridocyclitis**
    - It is quite common in the postoperative period. It is characterized by the presence of cells and flare in the anterior chamber. Sometimes, there may be an inflammatory membrane across the pupil due to inflammation
    - Incidence of iridocyclitis is higher in individuals with pre-existing uveitis, intraoperative excessive meddling of the iris, and incomplete removal of the cortex
    - They usually respond to corticosteroids and cycloplegics
    - Complications include pupillary membrane formation which leads to pupil block glaucoma, increased IOP due to clogging of trabecular meshwork by inflammatory cells

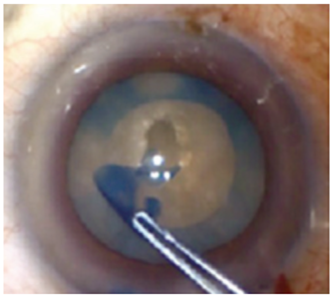
2. Have a careful look at the image shown below.

A. Identify the step of cataract surgery. (1 mark)

B. Name two ways in which this step can be performed. (2 marks)

C. Describe 7 postoperative complications of cataract surgery and their management. (7 marks)

A. Step is



Continuous curvilinear capsulorhexis.

B. Ways in Which this Step can be Performed

Capsulotomy can be performed by three techniques
1. Can-opener technique
2. Continuous curvilinear capsulorhexis

C. Complications

Postoperative Complications

They are divided into early and late postoperative complications

Early postoperative complications	Solution or treatment
1. Hyphema ▪ a collection of blood in the anterior chamber ▪ This can happen due to a bleed from the scleral wound or due to inadvertent iridodialysis	▪ It usually subsides and gets absorbed ▪ Treatment is supported with cycloplegics and topical corticosteroids ▪ If it is associated with raised intraocular pressure, antiglaucoma medications are given
2. Striate keratopathy ▪ It is the most common finding in the postoperative period and appears as lines of varying dimensions in the deep cornea due to folds along the Descemet's membrane. ▪ They may be dense if there was frequent shallowing of the anterior chamber intra-operatively	▪ Usually subsides over time

Contd.

<table>
<tr><th>Early postoperative complications</th><th>Solution or treatment</th></tr>
<tr><td>3. Corneal edema
They may be associated with dense striate keratopathy and occur as a result of endothelial damage or increased intraocular pressure</td><td>Topical hyperosmotic solutions are given to decrease corneal edema</td></tr>
<tr><td>4. Iridocyclitis
It is characterized by cells and flare in the anterior chamber</td><td>Topical steroids and cycloplegics</td></tr>
<tr><td>5. Acute postoperative endophthalmitis
It is a dreaded complication of any intraocular surgery
It is characterized by severe pain and diminished vision
On examination, there is lid edema, ciliary congestion, features of iridocyclitis, hypopyon, and a yellow reflex</td><td>Vitreous tap and aqueous tap
Intravitreal antibiotics
Vitrectomy</td></tr>
<tr><td>6. Iris prolapse
It is becoming rare with improved techniques of wound construction
It may present with in the first 24 hours or after a few days
The iris is found to prolapse through the wound and is associated with pupillary distortion
It forms a route of entry for organisms into the eye</td><td>If it is found within 6 hours of surgery, then the iris can be reposited back and the wound is sutured
If the duration is more, it is always safer to abscise the iris and suture the wound</td></tr>
<tr><td>7. Shallow anterior chamber
Causes are
<table><tr><td>Pupil block glaucoma</td><td>Wound leak</td></tr><tr><td>Malignant glaucoma</td><td>Ciliochoroidal detachment</td></tr><tr><td>Suprachoroidal hemorrhage</td><td></td></tr></table></td><td>Depends on the cause<table><tr><td>Pupillary block</td><td>Cycloplegics and steroids, Nd: YAG removal of the membrane</td></tr><tr><td>Malignant glaucoma</td><td>Cycloplegics
Vitrectomy</td></tr><tr><td>Suprachoroidal hemorrhage</td><td>Sclerotomy</td></tr><tr><td>Wound leak</td><td>Wound suturing</td></tr><tr><td>Ciliochoroidal detachment</td><td>Cycloplegics and steroids</td></tr></table></td></tr>
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SHORT ESSAYS

1. Explain the delayed postoperative complications following cataract surgery. (5 marks)

Delayed Postoperative Complications

1. **Cystoid macular edema**
    - The cause for this is inflammatory
    - Its incidence has come down drastically due to extracapsular surgery
    - The patient may complain of metamorphopsia, diminished vision, or scotoma

- Visual acuity is around 6/60–6/36
- On examination, one may see a honeycomb appearance at the macula
- It is confirmed by FFA (flower petal appearance due to leak of dye into cystoid spaces) and OCT (cystoid spaces in the inner retinal layers)
- Treatment is with topical NSAIDs, steroids, and periocular steroids

2. **Chronic delayed endophthalmitis**
   - It is due to organisms of low virulence which get entrapped in the capsular bag
   - They may be released following an Nd: YAG capsulotomy
3. **Pseudophakic bullous keratopathy**
   - This complication reflects the corneal endothelial damage that occurred during cataract surgery, especially if there were intraoperative complications
   - Risk factors include old age, low endothelial count, posterior capsule rent, vitreous in the anterior chamber, and use of ACIOL
   - As a result of damage to the endothelium, fluid enters the stroma and the cornea becomes opaque
   - Mild cases may resolve with hypertonic saline drops, but they usually require penetrating keratoplasty
4. **After cataract**
   - It is a complication seen only in extracapsular surgeries
   - It occurs due to the migration of epithelial cells from the pre-equatorial region to the posterior capsule to form an opacity
   - Predisposing factors include eccentric PCIOL (the haptic of the PCIOL prevents cell migration), postoperative iridocyclitis, and remaining cortical matter which could not be removed during surgery
   - The morphological types of after cataract is

	Description	Treatment
Sommering's ring	Thickening of the remaining capsular rim	It is usually in the periphery, hence does not need treatment
Elschnig's pearls	Soap bubble-like pearls appear beneath the capsule	If in the visual axis, Nd: YAG capsulotomy
Dense membranous after cataract	Dense thickening of posterior capsule	Surgical discission

5. **Retinal detachment**
   - May occur in high myopes, lattice degeneration, and more commonly seen in ICCE and associated vitreous loss during surgery
6. **Epithelial downgrowth**
   - Epithelium from the conjunctiva can grow into the anterior chamber through the incision and can lead to intractable glaucoma by covering the trabecular meshwork
   - It is a rare complication
7. **Fibrous ingrowth**
   - Growth of fibrous tissue into the anterior chamber, may cause glaucoma by a similar mechanism

2. Describe the intraoperative complications of cataract surgery. (5 marks)

Intraoperative Complications

Step	Complications	Solution
Superior rectus bridle suture	Laceration	Subsides on its own
Conjunctival flap creation	Excess bleed	Cauterization
Scleral incision	Too close to limbus—can cause premature entry into the anterior chamber Too far from limbus- difficult to maneuver	A viscoelastic substance can be injected to maintain the anterior chamber Another incision is made anteriorly if tunneling is not performed
Tunneling	Button-holing—too thin a flap can lead to the exit of the blade from anterior to the scleral incision Premature entry into the anterior chamber—it occurs due to deep dissection	Abandon this dissection and re-enter at a deeper plane from the other side of the external incision. Once detected, dissection should be stopped at that end and started at another end
During entry into the anterior chamber	Descemet detachment- it appears as a rolled edge at the entry of the wound, which may lead to postoperative corneal edema Injury to iris—iridodialysis	Subsides by itself Repair with sutures
Capsulotomy related	Too big a capsulorhexis leads to eccentric IOL placement Too small a capsulorhexis can lead to difficulty in delivering out the lens Run-away capsulorhexis—can lead to a posterior capsular tear	— It can be enlarged by raising another flap Stop at that point where the capsule is escaping and start on the opposite end
Hydro-dissection	A posterior capsular tear during forceful hydrodissection	Stop hydrodissection and use a vitrector to clear the vitreous and also the cortex.
Converting tunnel to funnel	Expulsive choroidal hemorrhage	Prevention by controlling IOP before surgery Sclerotomies to drain blood Form the anterior chamber and close with sutures Intravenous mannitol
Dialing out the nucleus	Posterior capsular tear if the nucleus is too big or capsulotomy is too small Zonular dehiscence may also happen	Identify the posterior capsular tear, do anterior vitrectomy and assess if the IOL can be placed in the capsular bag or anywhere else
Nucleus delivery	Damage to the endothelium Damage to the posterior capsule with wire Vectis is possible	Inflate the anterior chamber with a viscoelastic substance to prevent these. It coats the endothelium and helps in the smooth movement of instruments
Chopping the nucleus	Drop of the nucleus into vitreous due to posterior capsule rupture	Abandon the procedure, clear the cortex, form the anterior chamber performs a vitrectomy to remove the nucleus from the vitreous
Cortical wash and aspiration	Posterior capsular tear	Treat as explained in the hydrodissection associated complications
IOL insertion	Decentered IOL A tear in the posterior capsule	Depends on the extent of the tear in the posterior capsule

3. Outline the pros and cons of phacoemulsification vis-a-vis SICS. (5 marks)

Pros of Phacoemulsification- vis-à-vis SICS

1. Small incision—very low astigmatism, almost nil
2. Works with topical anesthesia
3. Postoperative congestion is minimal and recovery is faster
4. Less corneal complications—as the nucleus is not brought into the anterior chamber
5. Visual rehabilitation is faster

Cons of Phacoemulsification vis-à-vis SICS

1. High learning curve
2. Needs a machine, higher investment
3. Nucleus drop into vitreous is a dreaded complication
4. Higher cost
5. High phaco energy is needed to crack a hard lens

Pros of SICS over Phacoemulsification

1. Universal applicability to all types of cataracts
2. Easily performed in a nondilating pupil
3. Lesser surgical complications

Cons of SICS over Phacoemulsification

1. Postoperative pain is slightly more owing to the scleral incision.
2. More congestion
3. Delayed visual rehabilitation compared to phacoemulsification.
4. The incidence of hyphema is higher

4. Describe the steps of conventional ECCE. (5 marks)

- ECCE was the standard method of cataract extraction around 30–40 years back, where the posterior capsule was left behind and a posterior chamber IOL is implanted
- This type of surgery is almost obsolete now
- The steps of the same are
    - After painting and draping the eye with a cataract, a wire speculum is placed
    - Superior rectus bridle suture is applied
    - The superior fornix-based conjunctival flap is raised and episcleral vessels are cauterized
    - A scleral grove is made on the anterior limbus from 10 o'clock to 2 o'clock position
    - The anterior chamber is entered
- The anterior capsular opening is made (capsulotomy) by one of these three methods
    1. Can opener technique
    2. Continuous curvilinear capsulorhexis
    3. Envelope technique

- Hydrodissection
  - In this technique, a soft cannula is used to hydrate the cortex by irrigating between the margin of the anterior capsular opening and the cortex
  - By irrigation, the capsule gets separated from the nucleus and a majority of the cortex.
- The nucleus is then delivered out by using an irrigating wire Vectis or by pressure counter-pressure method
- The remaining cortex is washed with a Simcoe two-way irrigation aspiration cannula and removed.
- The capsular bag is injected with a viscoelastic substance and a PCIOL is implanted within the bag. And the remaining viscoelastic substance is washed off
- The anterior chamber is formed and the incision is sutured with 10-0 Nylon sutures.
- The conjunctival flap is reposited and the speculum is removed.
- The eye is closed and padded

5. ICCE was the choice of cataract surgery in the 1970s, however, it is obsolete now. Explain the indications of ICCE in the present day. Elaborate its steps. (1+4 marks)

- ICCE (intracapsular cataract extraction) is an obsolete technique today
- The only indication for ICCE is dislocation of the lens. As there is no zonular support left, the lens is removed in-toto

Steps of ICCE

1. After painting and draping the eye, a wire speculum is placed
2. A superior fornix-based conjunctival flap is raised to expose the sclera. Episcleral vessels are cauterized
3. An incision is made from 10 o'clock to 2 o'clock position at the superior limbus with a razor blade
4. The anterior chamber is entered with a keratome
5. Peripheral iridectomy is performed. Alpha chymotrypsin is used to dissolve the zonules
6. Methods of lens delivery
   - Cryoprobe is used to hold on the lens surface at a temperature of –40°C. the cryoprobe gets fixed to the lens. Then the probe is gently moved to and fro to loosen the zonules and the lens is delivered out
   - Wire Vectis method: Used to deliver out the lens in dislocation
   - Indian Smith method: In this method, the pressure is applied on the lower pole of the lens and counter pressure at 12 o'clock position and is brought out by the tumbling technique
   - Capsule holding method: In this technique, the anterior capsule is held with Arruga's capsule holding forceps, and gentle pressure is applied at the lower limbus to express the lens out
7. After the lens is delivered, the iris is reposited and an open sky vitrectomy is performed. Anterior chamber angle supported intraocular lens is introduced
8. The wound is sutured with 10–0 Nylon sutures and the conjunctival flap is reposited
9. Subconjunctival corticosteroid injection is given and the eye is padded

6. Explain the utility of various formulae in the calculation of IOL power. (5 marks)

SRK formula was the first of the formulae used for the calculation of the IOL power

First-generation formulae	SRK I formula	Most widely used formula Most suited for eyes in the range of 22.0-24.5 mm
Second generation formulae	SRK II formula	A is modified based on the axial length If L <20 mm: +3.0 is added If L is 20–20.99: +2.0 is added If L is 21–21.99: +1.0 is added If L is 22.0–24.5: 0 If L >24.5: –0.5
	Modified SRK II formula	A constant is also modified based on axial length
Third generation formulae	Holladay I and II formula	Uses average K, the radius of the cornea, required postoperative spherical equivalents, surgeon factor very exhaustive
	Hoffer's Q Haigis formula	Hoffer's Q is best suited for short eyes
Fourth-generation formulae	SRK/T formula	It is best suited for long eyes. Most accurate in the majority of cases Uses anterior chamber depth and retinal thickness
Universal formula	Barrett's formula	Can be used in all types of eyes including post-LASIK eyes

7. Explain the recent advances in cataract surgery. (5 marks)

- Cataract surgery has been involving from couching in the days of Hippocrates, through ICCE, ECCE, small incision cataract surgery, and now phacoemulsification
- With more and more sophistication being introduced, cataract surgery is being regarded as refractive surgery
- Cataract surgery has evolved in terms of instrumentation, surgical methods and also, the investigative techniques

Recent Advances

1. **Micro-incision cataract surgery or sleeve-less phaco**
   - This employs a co-axial system of irrigation aspiration thus reducing the overall diameter of the phaco probe
   - Thus the energy generated is less and the incision required is also smaller
2. **Femtosecond assisted cataract surgery**
   - Femtosecond LASER is used to create a tunnel in the cornea and also capsulorhexis so that the capsulotomy is flawless
   - The laser is also used to cause mild softening of the cataractous lens such that the amount of phaco energy used for nucleus removal is lesser
3. **Phaconit**
   - This technique uses a 1 mm-incsion to perform phacoemulsification

4. **Advances in intraocular lenses**
   - Multifocal IOLs, toric IOLs, and accommodating IOLs are being more commonly used that eliminate the use of presbyopic correction following cataract surgery
5. **Light-adjusted IOL**
   - It is made of photosensitive silicone macromer that gets polymerized on exposure ot light
   - After implantation, the eye is exposed to UV light based on the residual refractive power of the eye. unpolymerized macromer diffuses to the centre of the lens and swells according to the need of the refraction.
6. **Intraoperative aberrometry**
   - It gives surgeons more information on lens power and placement that helps in gaining 20/20 vision

8. Name four risk factors for posterior capsular tear? How is it recognized and managed? (2+3=5 marks)

Posterior Capsular Tear

Any breach in the continuity of the posterior capsule is defined as posterior capsular rent or tear.

Risk Factors

1. Morphology of cataracts: Black cataracts, posterior polar cataracts, and Morgagnian cataracts are more predisposed to posterior capsular tear
2. Traumatic cataracts
3. Long eyeball
4. Unstable capsulotomies and forceful hydrodissection

Recognition

- Sudden deepening of the anterior chamber with the movement of iris lens diaphragm posteriorly
- The pupil is dilated at the site of vitreous herniation
- Pupil snap sign: When a cotton bud is rolled at the wound if there is vitreous at the wound the corresponding part of the pupil moves
- The nucleus moves away from the phaco probe
- The edge of the posterior capsule may be seen against the red glow

Management

- PCT is small, with no disturbance of hyaloid face → dry aspiration of remaining cortex and implantation of PCIOL
- PCT >6 mm, or cannot visualize margins of the tear, then anterior vitrectomy → peripheral iridectomy → ACIOL
- PCT occurring in the early part of phacoemulsification (LIKE during capsulorhexis), then convert into small incision cataract surgery and deliver out the nucleus
- PCT with bits of nucleus remaining: viscoelastic is injected and nucleus pieces are brought out through an enlarged incision
- If the nucleus sinks into the vitreous, it should be removed using subtotal or total vitrectomy

9. Enumerate six early postoperative complications of cataract surgery. Explain the clinical features and management of anyone. (3+2 marks)

Early Postoperative Complications

1. Hyphema
2. Striate keratopathy
3. Corneal edema
4. Iridocyclitis
5. Glaucoma
6. Acute postoperative endophthalmitis
7. Shallow anterior chamber
8. Iris prolapse

Striate Keratopathy

Clinical Features

- Striate keratopathy is the most common postoperative complication
- It is characterized by folds in the Descemet's membrane due to sudden decompression of the anterior chamber
- The patients may report defective vision

Management

It subsides by itself and is supported by the use of topical steroids and cycloplegics.

10. Differentiate between toxic anterior segment syndrome (TASS) and acute postoperative endophthalmitis. (5 marks)

Feature	**TASS**	**Endophthalmitis**
Pathology	Sterile inflammation to irrigating solution or viscoelastic substance	Purulent inflammation due to bacterial
Timing	Immediate postoperative, 12–24 hours after surgery	It may take a few days to manifest, 2–7 days
Symptoms	Diminished vision	Pain is the most important differentiating feature
Lid edema	Uncommon	Always present
Corneal edema	Diffuse, limbus to limbus	Maybe sectoral
IOP	Extremely high, in the 50s	Unlikely to get a high IOP
Pupil	Dilated and fixed	Not necessarily
Response to topical steroids	Drastic, improvement within a day	No improvement
B scan	The posterior segment is clear	Exudates in vitreous

11. Name the causes for the postoperative shallow anterior chamber. Explain the diagnosis based on suitable clinical tests? (2+3 marks)

Causes for the Postoperative Shallow Anterior Chamber

It is seen either with a high IOP or a low IOP.

High IOP

1. Pupillary block
2. Malignant glaucoma
3. Suprachoroidal hemorrhage

Low IOP

1. Wound leak
2. Ciliochoroidal detachment

Investigating a Shallow Anterior Chamber with High IOP

- A good slit-lamp examination reveals a shallow anterior chamber that is deep at the center in the pupillary block. There is usually an inflammatory membrane
- In malignant glaucoma and suprachoroidal hemorrhage, there is a diffusely flat anterior chamber. However, in the latter, there is no red glow
- A B-scan shows kissing choroidal (dome-shaped detachment) with increased echogenicity

Investigating a Shallow Anterior Chamber with Low IOP

- A Seidel's test is performed by using a drop of freshly prepared fluorescein instilled in the conjunctival sac. The eye is examined under blue light
- A positive test is confirmed by the lightening of the dye at the site of the wound leak
- If Seidel's test is negative, then a B scan should be performed which shows dome-shaped choroidal detachment with low echogenicity

12. While performing cataract surgery on an 80-year-old, hypertensive, myopic, and long-standing glaucoma patient, the iris and the vitreous appear to be pushed out and the red glow is found to decrease.

A. What is the probable diagnosis? (1 mark)
B. Explain the risk factors and clinical features of this condition. (2 marks)
C. Explain the management of this condition. (2 marks)

A. Most Probable Diagnosis

Expulsive choroidal hemorrhage.

B. Risk Factors and Clinical Features

Risk Factors

- Old age
- Arteriosclerosis (long-standing hypertension)
- Long standing glaucoma
- Uncontrolled IOP prior to surgery

Clinical Features

Spontaneous gaping of the wound followed by the expulsion of the lens, vitreous, retina, uvea, and finally a gush of bright red blood.

C. Management

- Prevention by controlling IOP before surgery
- Sclerotomies to drain blood

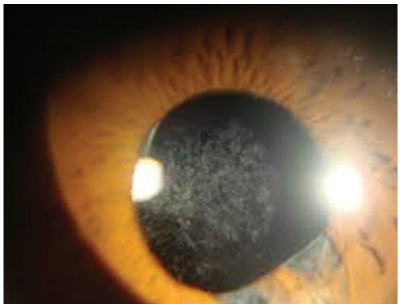
- Form the anterior chamber and close with sutures
- Intravenous mannitol
- Most of the time eye is lost and so evisceration should be performed

13. Mr G presents with diminution of vision 3 months after cataract surgery. It is gradual painless and progressive. slit-lamp examination shows the following image.

A. State the most probable diagnosis? (1 mark)

B. Name the clinical types. (2 marks)

C. Explain its management. (2 marks)



A. Most Probable Diagnosis

After-cataract

B and C. Clinical Types and Management

Clinical type	Description	Management
Sommering's ring	Thickening of the remaining capsular rim	It is usually in the periphery, hence does not need treatment
Elschnig's pearls	Soap bubble-like pearls appear beneath the capsule	If in the visual axis, Nd: YAG capsulotomy
Dense membranous after cataract	Dense thickening of posterior capsule	Surgical discission

SHORT ANSWERS

1. Compare small incision cataract surgery (SICS) and extracapsular cataract extraction (ECCE). (3 marks)

Comparison of SICS and ECCE

Feature	SICS	ECCE
Size of the incision	6–7 mm	10–12 mm
Degree of postoperative astigmatism	A lesser degree of astigmatism	Longer incision, higher astigmatism
Suture	No need for sutures and thus no suture-related complications like irritation, an abscess	Postoperative suture-related problems are higher
Healing time	Smaller incision, faster healing	Longer incision, longer time to heal
Postoperative complications	Fewer chances of postoperative shallowing of the anterior chamber Fewer chances of vitreous-related complications	Higher incidence of wound leak, iris prolapse, and shallowing of the anterior chamber

2. Explain the use of biometry in cataract surgery. (3 marks)

- Biometry is the calculation of the power of the IOL or intraocular lens that is to be placed instead of the cataractous human crystalline lens following its extraction
- This power is individualized based on each patient's corneal curvature, axial length, and the effective position of the lens

- Hence, a formula was developed to calculate IOL power by Sanders-Retzlaff, and Krauff (SRK formula) which is

$$\text{Power} = A - 0.9K - 2.5L$$

  Where,
  A = constant for the material of the IOL used
  K is the mean of vertical and horizontal anterior corneal curvature in diopters (measured by keratometer)
  L is the axial length of the eyeball in mm (measured by A-scan ultrasound)
- However, many newer versions have been developed for each type of eye like SRK II, Haigis and others
- It is imperative before each cataract surgery and each eye. The power of the IOL calculated is kept ready before the surgery

3. Explain the clinical features of floppy iris syndrome. (3 marks)

- Floppy iris syndrome is a condition in which the iris becomes flaccid intraoperatively during cataract extraction
- The main etiology is supposed to be systemic use of Tamsulosin ($\alpha 2$ antagonist) for bladder outlet symptoms
- Preoperative identification is usually only a poorly dilating pupil
- Intraoperatively, floppy iris syndrome is diagnosed by a triad of features
  1. The billowing of the iris with an infusion of fluid into the anterior chamber
  2. The propensity of the iris to prolapse from the incisions and side ports
  3. Progressive constriction of the pupil with surgery
- Management includes the use of viscoelastic and use of iris hooks to keep the pupil dilated throughout the surgery

4. What are the implications of pseudoexfoliation syndrome in cataract surgery? (3 marks)

- Pseudoexfoliation syndrome is a fairly common finding characterized by dandruff-like powder seen on the anterior capsule of the lens
- There is a higher incidence of cataract formation, especially nuclear cataracts in patients with pseudoexfoliation
  - The pupil is usually poorly dilating which makes the surgery difficult. The capsulorhexis may be difficult to perform
  - There is a higher incidence of glaucoma in patients with pseudoexfoliation
  - Also, there is some amount of zonular weakness. So, the surgeon should be careful during nucleus delivery

5. Explain why the use of ACIOL is on the decline. (3 marks)

- ACIOL is mainly indicated in absence of zonular support as in following an ICCE
- The haptics of the IOL is supported at the angle. However, it is not physiological and not free of complications
  - UGH syndrome is a unique triad of uveitis, glaucoma, and recurrent hyphema
    - It occurs due to frequent rubbing of the IOL against the iris, causing uveitis and glaucoma
    - Hyphema may occur due to erosion of blood vessels on the surface of the iris and in the angle
  - These patients sometimes may develop endothelial decompensation

- There are better options available such as iris-claw lenses and scleral fixated IOLs which are more convenient for the patient and more physiological

6. A 69-year-old patient with cataract complains of severe pain during a retrobulbar anesthesia.On examination, there is proptosis, conjunctival hemorrhage, chemois, and restricted ocular movements. The eye feels hard.

A. What is the most probable cause? (1 marks)

B. Explain the management of the same. (2 marks)

A. Most Probable Diagnosis

- Retrobulbar hemorrhage: It may occur due to inadvertent damage to vessels in the retrobulbar space during retrobulbar anesthesia

B. Management

- It is treated by ocular massage
- If it does not subside, the eye has to be closed and padded and intravenous mannitol or acetazolamide has to be given to decrease IOP to avoid ischemia of the central retinal vessels
- Lateral canthotomy is performed to relieve the orbital pressure

7. Explain the importance of a good capsulorhexis in cataract surgery. (3 marks)

- Capsulorhexis is an important step in any type of cataract surgery and is especially important in phacoemulsification
- The size of the rhexis should be enough to perform the nucleus chop and aspiration. The ideal size of rhexis is 5.5 mm in diameter
- Too small a rhexis can lead to difficulty to deliver the nucleus. Too large a rhexis can lead to an eccentric positioning of the IOL
- The capsulorhexis margins can sometimes run away and lead to a posterior capsular tear. This hampers adequate positioning of the IOL

8. Recognize the signs of vitreous loss during cataract surgery. (3 marks)

Signs of Posterior Capsule Rupture and Vitreous Loss

- Anterior chamber deepens suddenly
- Movement of iris lens diaphragm posteriorly
- The pupil is dilated at the site of vitreous herniation
- Pupil snap sign—when a cotton tipped applictaor is rolled at the wound if there is vitreous at the wound the corresponding part of the pupil moves
- The nucleus moves away from the phaco probe

9. Explain the clinical features and management of postoperative cystoid macular edema. (3 marks)

Postoperative Cystoid Macular Edema

Clinical Features

- It characteristically appears 4–6 weeks after cataract extraction
- The patient may complain of metamorphopsia, diminished vision, or scotoma
- Visual acuity is around 6/60–6/36

- On examination, one may see a honeycomb appearance at the macula
- It is confirmed by FFA (flower petal appearance due to leak of dye into cystoid spaces) and OCT (cystoid spaces in the inner retinal layers)

Management

- Topical NSAIDs
- Topical steroids
- Periocular steroids

10. Enumerate IOL-related complications. (3 marks)

IOL-related Complications

1. UGH syndrome: It is usually caused due to anterior chamber IOLs which rub on the iris and lead to recurrent episodes of uveitis, glaucoma, and hyphaema
2. Decentered IOL: It may cause distorted vision or diplopia
3. Wrong IOL: It must be exchanged

COMPETENCY

OP7.5 TO PARTICIPATE IN THE TEAM FOR CATARACT SURGERY

SHORT ANSWERS

1. Explain the preoperative preparation of a patient with a cataract. (3 marks)

- A patient that has been selected for cataract surgery after a careful evaluation is preferably given a dose of topical antibiotics for 24 hours
- There should be control of IOP, blood sugars, and blood pressure using suitable drugs if needed
- Some patients may be anxious, and so must be given anxiolytics on the previous night
- If the intended procedure is to be performed under sedation or general anesthesia, the orders given by the anesthetist should be followed
- A xylocaine sensitivity test is performed on the forearm

2. Explain the procedure of peribulbar anesthesia. (3 marks)

- Peribulbar anesthesia is a type of regional anesthesia where the local anesthetic is delivered into the peripheral space of the orbit using a 24-gauge needle
- The patient is made to lie down comfortably in a supine position
- The eye is cleaned, and topical anesthetic (proparacaine) is instilled. The skin around the eye is cleaned with spirit or povidone-iodine
- The patient is instructed to look straight up
- The lower orbital margin is felt with the nondominant hand and a spot at the junction of lateral thirds and medial two-thirds is noted
- The syringe containing lignocaine 2% + 1:1,00,000 + 5IU/ml of hyaluronidase is taken with a needle of 24 gauge bore and 1½ inch length. It is inserted vertically, and the eye movements are tested to rule out accidental insertion into a muscle or the globe
- Around 4–5 ml of solution is injected followed by an ocular massage for 5–10 minutes
- If the movements are persisting, an additional block of not more than 2 ml can be given at the supraorbital notch

3. Explain how phacoemulsification can be performed under topical anesthesia. (3 marks)

- Phacoemulsification is performed through a corneal incision in the peripheral cornea
- Topical anesthesia is administered by proparacaine, and the patient is instructed to not move their eye as much as possible
- As the scleral incision is not made, phacoemulsification can be performed under topical anesthesia

4. Explain the necessity of maintaining a sterile atmosphere in an ophthalmic operating room. (3 marks)

- Intraocular procedures need utmost precautions concerning sterility and disinfection
- Even though the postoperative infection is of low incidence, it has devastating visual sequelae
- A sterile atmosphere is maintained by limiting the number of individuals in the operating room, cleaning and disinfecting the room after every procedure, use of a new set of sterile instruments, and having clean air inside the room
- The quality of air in the operating room is tested using blood agar placed next to the operating table
- Appropriate sterilization methods should be employed for objects

COMPETENCY

OP7.6 ADMINISTER INFORMED CONSENT AND COUNSEL PATIENTS FOR CATARACT SURGERY IN A SIMULATED ENVIRONMENT

SHORT ESSAY

1. A 75-year-old patient was being given retrobulbar anesthesia before cataract surgery. He suddenly feels dizzy, and his pulse rate is 56/min.

A. State the most probable explanation for this phenomenon. (1 mark)

B. Explain the pathway and features of the same. (2+2 marks)

A. Most Probable Cause for This

- Oculocardiac reflex
- It is also known as the Aschner reflex

B. Pathway

- It is a trigemino-vagal reflex
- When there is stretching of the extraocular muscle or globe compression, there is vagal stimulation that leads to sudden bradycardia and sometimes death
- It is diagnosed when the heart rate drops >20%
- It is an expected complication during retrobulbar anesthesia
- The stretch receptors are stimulated during anesthesia cause stimulation of the vagal nucleus that leads to bradycardia (Fig. 7.6.1)

Clinical Features

- Th reflex may be seen during peribulbar anesthesia and squint surgeries
- This reflex is also stimulated during a blow-out fracture when a muscle belly is caught in the fracture site
- The patient may develop sinus bradycardia, asystole, lightheadedness, nausea, weakness, arrhythmias, or even death

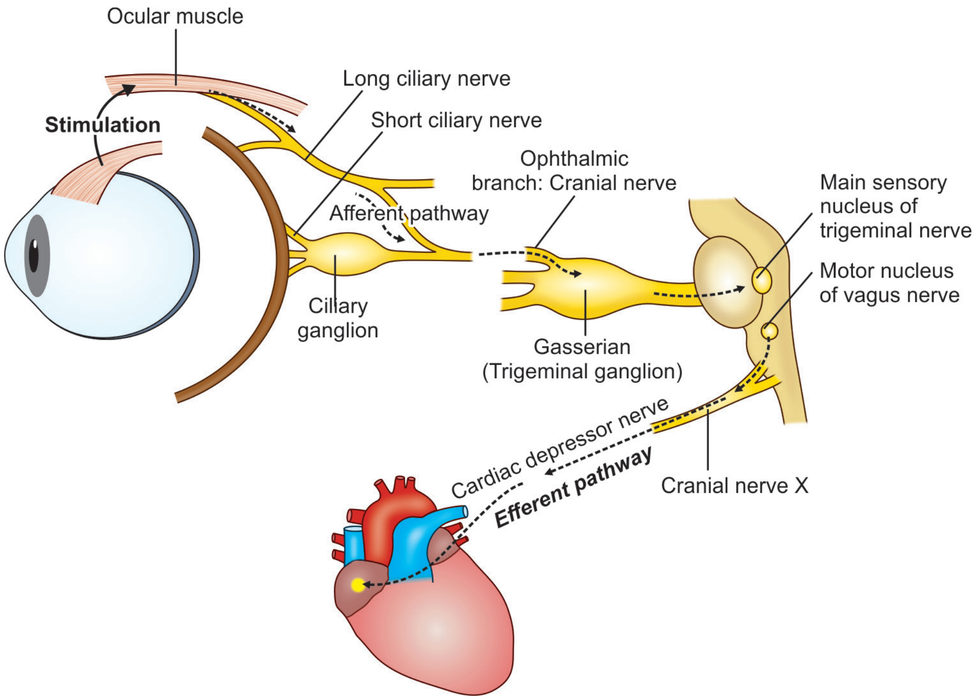

Ocular muscle
Stimulation
Long ciliary nerve
Short ciliary nerve
Ophthalmic branch: Cranial nerve
Afferent pathway
Ciliary ganglion
Main sensory nucleus of trigeminal nerve
Motor nucleus of vagus nerve
Gasserian (Trigeminal ganglion)
Cardiac depressor nerve
Efferent pathway
Cranial nerve X

Fig. 7.6.1: Pathway of oculocardiac reflex

SHORT ANSWERS

1. Identify the indications of cataract surgery. (3 marks)

- The earliest and most consistent symptom is defective vision. Any cataract that interferes with the day-to-day activity of a patient necessitates removal by surgery
- In the pediatric age group, any cataract in the visual axis or more than 3 mm in diameter should be indicated for cataract surgery
- Other indications are complications of cataracts like phacolytic and phacomorphic glaucoma.
- Primary angle-closure glaucoma is also being treated by cataract extraction as it eliminates pupillary blocks
- A refractive indication is a very high ametropia that is treated by refractive lens exchange

2. Explain the situations when the patient is informed about poor visual prognosis following cataract extraction. (3 marks)

- Cataract surgery is one of the most rewarding surgeries as the visual prognosis is excellent
- Nonetheless, poor visual prognosis following cataract surgery is expected in many special situations
- They are
  1. **Macular disorders**
     - Various macular disorders may be present along with a cataract like diabetic macular edema, dystrophies, age-related macular degeneration, epiretinal membrane, and so on
     - The health of the macula must be tested before a cataract surgery using appropriate macular function tests

2. **Traumatic cataract**
   - Some cases of traumatic cataracts may be associated with permanent posterior segment pathologies
3. **Associated ocular disorders**
   - Cataracts may be associated with advanced glaucoma and field defects. These defects are irreversible and their nature should always be conveyed to the patient
   - Corneal dystrophies, opacities, may interfere with visual acuity
   - Neuro-ophthalmic conditions like partial optic atrophy may be a cause for decreased vision along with cataracts
   - Phacomorphic and phacolytic glaucoma: there may damage to the optic nerve due to raised intraocular pressure
   - Complicated cataract: Associated with uveitis or retinitis pigmentosa
4. **Nonvisualization of fundus**
   - Sometimes, a dense cataract may not allow fundus assessment
   - Also, if macular function tests show negative results, the patients must be warned of a poor visual outcome
5. **Possible amblyopia**
   - This is important in the case of pediatric cataracts when the cataract has not been operated on for a very long time during the amblyogenic period
   - Also, when the visual loss is much more than the amount of cataract, amblyopia may be suspected.

CHAPTER

8

Retina and Optic Nerve

COMPETENCY

OP8.1 DISCUSS THE AETIOLOGY, PATHOLOGY, CLINICAL FEATURES AND MANAGEMENT OF VASCULAR OCCLUSIONS OF THE RETINA

LONG ESSAYS

1. A 66-year-old man with hypertension, diabetes, and hyperlipidemia presents with episodes of sudden obscuration of vision lasting for 20–30 seconds, followed by complete recovery.

A. What is this symptom called? (1 mark)

B. What is its premonitory sign? (1 mark)

C. Classify vascular occlusion disorders of the eye with underlying etiologies. (5 marks)

D. Contrast between arterial and venous occlusion. (3 marks)

A. Symptom Called

This symptom of transient obscuration of vision lasting for 20–30 seconds followed by complete recovery is called Amaurosis fugax.

B. Premonitory Sign

- Central retinal artery occlusion
- However, it can also be seen in papilledema

C. Classification of Vascular Occlusions of the Eye

Arterial Occlusion

1. Precapillary retinal arteriole obstruction
2. Branch retinal artery occlusion (BRAO)
3. Central retinal artery occlusion (CRAO)
4. Ocular ischemic syndrome
5. Ophthalmic artery occlusion

Venous Occlusion

1. Branch retinal vein occlusion (BRVO)
2. Central retinal vein occlusion (CRVO)
   i. Ischemic CRVO
   ii. Nonischemic CRVO
3. Macular branch venous occlusion

Etiology of Arterial Occlusive Disease

Extraluminal Causes

- A sudden rise in intraocular pressure more than the pressure in the central retinal artery (~60 mm Hg) can be due to
  - Acute congestive glaucoma
  - Following intravitreal injection
  - Any other forms of glaucoma
- Compression on the optic nerve after entry of the central retinal artery (within 1 cm posterior to lamina cribrosa)
  - Orbital compartment syndrome due to emphysema or hemorrhage
  - Traumatic injury optic nerve avulsion
  - Orbital tumors—sudden expansion due to hemorrhage

Intraluminal Causes

Complete obstruction of the lumen of the central retinal artery by

- Atherosclerotic thrombi at lamina cribrosa (80% of causes)
- Carotid emboli—usually arise at the bifurcation, and since the left internal carotid has a straighter course, left CRAO is more common due to embolic phenomena
  - Calcific emboli—from calcified valves. They are very dangerous, as they cause permanent obstruction
  - Fibrin platelet—gray appearing, lead to amaurosis fugax and then complete obstruction. They are usually multiple
  - Cholesterol emboli—Hollen horst plaques, rarely cause obstruction but appear as golden colored plaques within the arterioles

Rare Causes

- Giant cell arteritis
- Vegetations from bacterial endocarditis
- Inflammatory periarteritis as in SLE, PAN, Wegener's granulomatosis, and Behcet's syndrome
- Thrombophilic disorders
- Sickle cell anemia
- Retinal migraine
- Myxomatous cells from myxomas

Etiology of Venous Occlusive Disease

- Arteriosclerosis of the retinal artery may lead to atherosclerosis of the retinal vein as they share the same common adventitious sheath, thickening of the artery may lead to hemodynamic changes in the veins
- Predisposing factors include advancing age, hypertension, diabetes mellitus, Hyperlipidemia, and open-angle glaucoma

Rare Causes

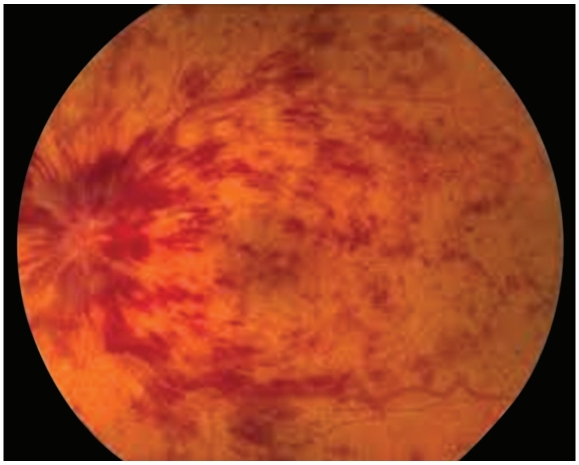
- Myeloproliferative disorders like polycythemia, abnormal plasma proteins
- Hypercoagulable states like homocystinemia, APLA, dysfibrinogenemia
- Hereditary hypercoagulable conditions like protein C deficiency, protein S deficiency, antithrombin, and prothrombin deficiency
- Collagen vascular diseases
- Oral contraceptives and hormone replacement therapy

D. Differences between Arterial and Venous Occlusions

Both conditions present with sudden painless diminution of vision

Feature	Arterial occlusions	Venous occlusion
Mechanism of occlusion	Embolic and thrombotic phenomena	Thrombotic phenomena Increased viscosity of blood
Appearance of retina	Pale due to total ischemia	Filled with hemorrhages due to rupture of capillaries
Blood vessels	Arteries are severely narrowed	Venous dilatation and tortuosity
Risk of developing neovascular glaucoma	Lower in arterial occlusion	Higher in venous occlusion
Visual prognosis	Poor	Moderate to poor
Type of optic atrophy	Primary optic atrophy	Secondary optic atrophy

2. A 50-year-old man presents with sudden painless diminution of vision. Fundus examination shows the following image.



A. What is the most probable diagnosis? (1 mark)
B. Differentiate between the two main variants. (3 marks)
C. Explain the management of this condition. (3 marks)
D. List three systemic associations for bilateral affection (3 marks)

A. Most Probable Diagnosis

Ischemic central retinal vein occlusion

B. Differences between Ischemic and Nonischemic CRVO

Ischemic and Nonischemic CRVO

Parameter	Nonischemic	Ischemic
Visual acuity	Usually >6/60	Usually <6/60
Pupil	RAPD may or may not be there	Always present
Disc	No edema	Edematous
Hemorrhages	Less	More
Areas of non-perfusion (on FFA)	<10 DD	>10DD
ERG	b-wave amplitude >60% normal	b-wave amplitude <60% normal
Prognosis	Fewer chances	Higher chances of developing neovascular glaucoma

C. Management

- Any systemic association like hypertension, diabetes, hyperlipidemia, and glaucoma should be looked for and treated or controlled
- The hemorrhages resolve over time and do not need any immediate treatment. The patient needs to be followed up frequently
- Treatment of macular edema
  - It usually does not benefit from macular LASER
  - It may benefit from intravitreal triamcinolone and anti-VEGF medications like ranibizumab, bevacizumab
- '100 day' glaucoma or neovascular glaucoma
  - The patient is reviewed every three months and a gonioscopy should be performed to look for any new vessels in the angle
- To prevent neovascularization
  - LASER photocoagulation to be given when at least two clock hours of iris show new vessel formation.
  - Intravitreal anti-VEGF drugs are becoming the drug of choice these days
- Surgical methods: Optic nerve sheathotomy and intravenous tissue plasminogen are being tried

D. Systemic Associations for Bilateral CRVO

1. Multiple myeloma
2. Homocystinemia
3. Use of oral contraceptives

SHORT ESSAYS

1. Annotate the arterial supply of the retina with its unique properties. (5 marks)

The majority of the arterial supply of the retina is through the central retinal artery except for a small peripapillary area which is supplied by short posterior ciliary arteries and the choroid.

Central Retinal Artery

- The central retinal artery, a branch of the ophthalmic artery, is an end artery and it enters the globe along with the optic nerve
- It pierces the dura at 1 cm behind the lamina cribrosa and contains a tunica intima (consisting of a single layer of endothelium), internal elastic lamina, tunica media (consists of smooth muscle), and tunica adventitia

Branches

- The central retinal artery branches into a superior and an inferior branch which then further branch into a nasal and a temporal branch
- Thus, four major arterial arcades are arising from the central retinal artery at the level of the optic disc
- Then, they divide dichotomously and end in capillaries

Capillaries

- The capillaries have endothelial cells lined on a basement membrane and a cuff of pericytes
- The tight junctions between the endothelial cells from the blood–retinal barrier
- They supply the inner two-thirds of the retina, whereas the outer third of the retina is supplied by the choriocapillaris
- They lie in the inner nuclear layer and the ganglion cells layer. Whereas in the macula, they are much denser and are four layers of capillary complexes
- The parts of the retina that are free of capillaries are:
  - Perivascular areas
  - Foveal avascular zone
    - Derives its nutrition from the underlying choriocapillaris
    - It is around 500 μm

Short Posterior Ciliary Arteries

They arise as 15–20 branches off the ophthalmic artery and mainly supply the optic nerve head and peripapillary choroid.

Choroidal Vessels

The choriocapillaris supply the outer 4 layers of the retina.

Unique Properties of Retinal Circulation

- The horizontal raphe serves as a watershed zone
  - The arteries or veins never cross the horizontal raphe
  - In cases of long-standing branch arterial obstruction, there can be collaterals across the horizontal raphe
- Since these are end arteries, any block in the vessels leads to severe ischemia of the area supplied by these arterioles
- Blood–retinal barrier is formed by the tight junctions between the capillaries which prohibit the free flow of fluids across the tight junctions
- The pericytes have contractile properties and are associated with autoregulation. Thus, systemic blood pressure does not affect the perfusion of the retina
- The macula is supplied by the cilioretinal artery in a variable number of eyes and ranges from 6–30% in each population
  - It arises from the ciliary circulation
  - So, in cases of central retinal artery occlusion, there is profound ischemia of the entire retina, leaving out a well-perfused macula, and hence preserving some functional vision

2. Describe the etiology and clinical presentations of central retinal artery occlusion. (3+2 marks)

Central Retinal Artery Occlusion

Etiology

Refer to page No. 312, Q. No. 1 of Long Essay.

Clinical Features

- Patients present with unilateral sudden painless loss of vision which may seem like a curtain dropping in front of the affected eye. It may be preceded by episodes of transient obscuration of vision
- CRAO associated with giant cell arteritis may complain of severe one-sided headache, jaw claudication. Similarly, patients with migraines may also complain of headaches
- Examination findings show:
  - Severely diminished vision (hand movements or perception of light only)
  - Afferent pupillary defect
  - Fundus shows diffuse whitening (due to edema) of the retina with arterial narrowing and may show a segmented column of blood due to stagnation of flow. This is called the cattle trucking phenomenon
  - The fovea appears reddish-brown due to the underlying choroid and is called 'Cherry red spot'
  - If there is a cilioretinal artery present, the area supplied by it (papillomacular bundle) stands out against the surrounding pale retina and the vision is 6/6 with a severely contracted visual field
  - After around 6-8 weeks, cherry red sot and cloudiness disappear, and consecutive optic atrophy develops
  - Various types of thrombi/emboli may be seen in the vessels

3. A 65-year-old man presents with sudden painless loss of vision in his right eye noticed 2 hours back. On examination, there is RAPD and a cherry-red spot in the retina with surrounding retinal pallor. Explain the management of an acute episode of this condition. (5 marks)

The most probable diagnosis is central retinal artery occlusion (CRAO)

Evaluation

- Patients with CRAO must be evaluated as they almost always have an underlying systemic condition
- They must be investigated thoroughly for systemic abnormalities as they may be life-threatening
- Systemic evaluation should include
- Pulse—to detect atrial fibrillation
  - Blood pressure
  - Carotid evaluation for detecting bruit and doppler ultrasound
  - ECG and echocardiography
  - Blood investigations for ESR, blood count, sickling, homocysteine, and ANA
  - Thrombophilic screen
- CRAO can be reversed if the patient presents very early (<2–3 hours of presentation)

Emergency Treatment (within 2–3 hours of loss of vision)

- Immediate lowering of IOP leads to dislodgement of the thrombus.
    - Ocular massage—with the ball of the thumb for 10 seconds followed by a release for 5 seconds. It can be even done with a gonio lens
    - Oral or intravenous acetazolamide
    - Anterior chamber paracentesis
- Inhalation of carbogen (95% oxygen + 5% CO_2) by breathing in a closed bag
- Sublingual glyceryl trinitrate—to relieve the angiospasm

To Prevent Further Attacks and Systemic Treatment

- Anticoagulants
- Systemic steroids for collagen vascular diseases
- Carotid artery endarterectomy if the obstruction is more than 90%

4. Describe the etiology, clinical features, and management of the ocular ischemic syndrome. (2+2+1= 5 marks)

Ocular Ischemic Syndrome

Etiology

- It results due to chronic hypoperfusion of the eye due to atherosclerotic thrombotic obstruction of the ipsilateral carotid artery (>90% luminal obstruction, >50% reduction in perfusion) caused by atherosclerosis, Eisenmenger syndrome, giant cell arteritis, etc.
- It is associated with diabetes, ischemic heart disease, hypertension, and cerebrovascular disease
- It is usually seen in individuals older than 55 years of age

Clinical Features

Symptoms
- A gradual decrease in vision
- May be associated with ocular pain.
- It may be preceded by amaurosis fugax (episodes of transient obscuration of vision)

Signs
- Ciliary congestion
- Corneal edema
- Anterior chamber: cells, flare (ischemic pseudo-iritis)
- Iris shows atrophic patches, mid dilated poorly reacting pupil
- Rubeosis iridis may occur and develop neovascular glaucoma
- Cataract in advanced cases
- Fundus shows venous dilatation, arterial narrowing, hemorrhages, and occasional disc edema. Proliferative retinopathy develops due to chronic ischemia

Management

- Anterior segment: Topical steroids and mydriatics
- Neovascular glaucoma: Medically or surgically depending on the response
- Antiplatelet therapy
- Carotid stenting and endarterectomy

5. A 32-year-old man presents with bilateral central retinal vein occlusion. Enlist the investigations that will help to recognize rare causes of central retinal vein occlusion in this case.

- Since the age of presentation is very early and also, it is a bilateral affection, systemic causes should be looked for
- These are:

Investigation	Probable pathology
Blood pressure Renal doppler serum catecholamines	Malignant hypertension
Lipid profile	Familial hypercholesterolemia
ANA profile RA factor ANCA Anticardiolipin antibodies	Collagen vascular disease
ESR chest X ray Quantiferon gamma VDRL	Infectious vascultidis (tuberculosis, syphilis)
Peripheral blood smear Bone marrow aspiration and biopsy	Myeloproliferative disorders
Serum homocysteine levels	Homocysteinemia
Protein C Protein S Antithrombin III levels Factor V Leiden mutation	Hereditary hypercoagulable states
Bence Jones proteins M band in serum electrophoresis	Multiple myeloma
Polyclonal Ig M antibodies	Cryoglobulinemia
Packed cell volume	Intense exercise causing dehydration

6. Describe the clinical features and management of branch retinal vein occlusion. (3+2= 5 marks)

Retinal Vein Occlusion

Clinical Features

It can be of three types:

1. Major BRVO
2. Macular BRVO
3. Peripheral BRVO
    - It is more common than CRVO
    - Usually, superotemporal branch retinal vein occlusion is the most common type of the lot

Symptoms

- Superotemporal and macular BRVO presents with sudden, painless blurring of vision
- The reason is that they are always symptomatic due to macular edema that occurs due to the gravitation of fluid
- Other types of BRVO remain asymptomatic

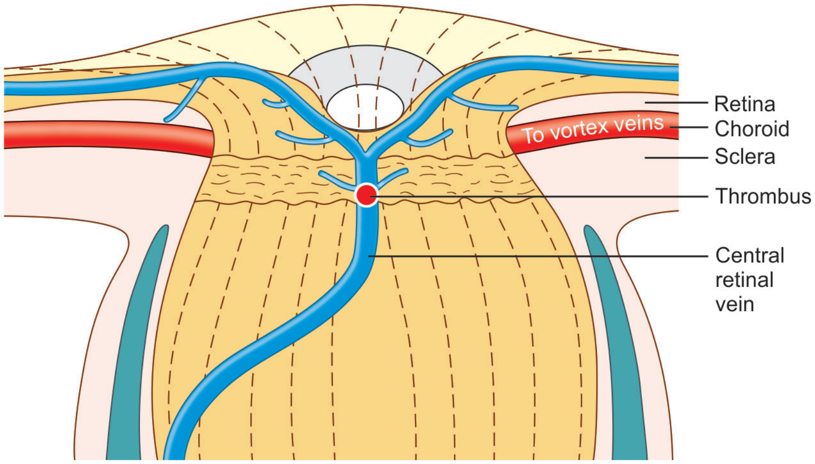
Signs

- Visual acuity depends on the amount of macular ischemia
- RAPD may or may not be present
- Fundus examination shows superficial flame-shaped hemorrhages scattered along the course of the involved arcade, along with dot-blot hemorrhages and cotton wool spots
- After 6–9 months, there is sheathing, hard exudates, variable number of residual hemorrhages
- There are collaterals within the retina, and they are seen crossing the horizontal raphe

Management

- FFA is done usually 6–12 weeks after the occlusion has occurred, provided the hemorrhages have resolved
- If macular perfusion is good and visual acuity has improved, there is no need for any treatment
- If macular perfusion is good, but vision is 6/12 or worse even after 3 months, then grid photocoagulation is done to occlude the leaky vessels
- If macular perfusion is poor (FAZ >500 mic), the visual prognosis is bad. Intravitreal triamcinolone may improve some visual acuity
- If FFA shows >5 disc areas of non-perfusion risk of neovascularization is high. The patient is followed up every 3 months with gonioscopy and dilated fundus examination. If new vessels are seen, a scatter laser is given

7. If a thrombus were to completely obstruct the site as shown in the image, summarize the likely complications and their management in this patient?



Or

Describe the complications of central retinal vein occlusion and their management. (3+2 marks)

- A thrombus that completely occludes the central retinal vein at the lamina cribrosa causs ischemic central retinal vein occlusion
- The complications arise due to the widespread ischemia of the retina
- As a result, VEGF is produced by the retinal cells that cause neovascularization

Complication	Management
Neovascular glaucoma • It occurs in about 20% of patients with CRVO and is more common in the ischemic variant as the extent of ischemia is greater • It usually occurs after 3 months and the incidence is higher if the FFA shows capillary drop-out areas of more than 5DD • Neovascular glaucoma is screened for by gonioscopy and dilated fundus examination at regular intervals	• According to the CVOS (central venous occlusion study), iris neovascularization >2 clock hours is the indication for pan-retinal photocoagulation. Intravitreal anti-VEGF agents can also be used • This helps to delay the occurrence of neovascular glaucoma • Once glaucoma develops, antiglaucoma medications, topical cycloplegics are used. It is followed by glaucoma drainage devices and cyclodestructive procedures
Vitreous hemorrhage • It develops due to VEGF-induced neovascularization. The new vessels are extremely delicate and tend to bleed • These also form fibrovascular membranes that can cause tractional retinal detachment	• It is treated with vitrectomy, endophoto-coagulation, and reattachment of the retina
Macular edema • It is a major cause of decreased visual acuity, especially in the nonischemic variant • It occurs due to ischemia and due to fluid transudation into various layers of the macula	• Grid photocoagulation decreases macular edema but has not been found to improve visual acuity • Intravitreal anti-VEG-F agents and steroids
Optic atrophy • It is more of a sequel • It is characterized by the dirty yellow pallor of the disc associated with the sheathing of blood vessels • There are collaterals around the disc	• There is no treatment once optic atrophy ensues

8. Explain the clinical features and management of papillophlebitis. (3+2 marks)

Papillophlebitis

- An inflammatory variant of central retinal vein occlusion
- Also, known as optic disc vasculitis

Clinical Features

Symptoms

- It is usually seen in a younger population
- They present with unilateral or bilateral painless blurring of vision

Signs

- There is inflammation of the disc and resultant venous congestion
- Hemorrhages are usually confined to the peripapillary region
- The amount of disc edema is out of proportion to the number of hemorrhages
- RAPD is always present and blind spot is enlarged on visual field analysis
- The visual prognosis is good

Management

- Systemic corticosteroids
- Anticoagulants
- Intravitreal anti-VEGF agents can be tried in cases of severe macular edema
- If not treated promptly, may progress to ischemic CRVO

SHORT ANSWERS

1. Enumerate three differential diagnosis of a cherry-red spot. (3 marks)

1. Central retinal artery occlusion
2. Berlin's edema
3. Sphingolipidosis—Gaucher's disease, Niemann–Pick disease, Tay–Sachs disease
4. Macular hole with surrounding serous retinal detachment
5. Quinine toxicity

2. Explain the pathogenesis of central retinal venous occlusion. (3 marks)

- Arteriosclerosis of the retinal artery may lead to atherosclerosis of the retinal vein
- As they share the same common adventitious sheath, thickening of the artery may lead to hemodynamic changes in the veins
- Similarly, even the central retinal artery and vein share a common adventitious sheath, just posterior to the lamina cribrosa
- A complete occlusion gives rise to the ischemic variant of the CRVO and partial obstruction causes a nonischemic variant of the CRVO
- It can also be caused when the intraocular pressure is high enough to occlude the central retinal vein

3. Describe the clinical features of macular branch retinal vein occlusion. (3 marks)

- Macular branch vein occlusion is caused by occlusion of the venule draining the macular area
- It affects the superior macular vein more commonly than the inferior macular vein
- These patients present with a central field defect and metamorphopsia
- There are numerous flame-shaped hemorrhages seen in the macular area only, with venous dilatation and tortuosity proximal to the occlusion
- There may be a few cotton wool spots
- There is also associated macular edema

4. Explain the role of fluorescein angiography in the management of retinal venous occlusion. (3 marks)

- Fluorescein angiography is a technique of visualizing the vasculature of the retina and partly, the choroid after injecting a hydrophilic agent called fluorescein
- In cases of acute central retinal venous occlusion, the vasculature cannot be studied due to concealment of the fluorescence by the hemorrhages (blocked fluorescence)
- When performed after 6-8 weeks, it allows recognition of capillary nonperfusion areas.
  - If the total of capillary nonperfusion areas is more than 5 DD in size, there is a high chance of the development of neovascular glaucoma.
  - Such cases should receive laser photocoagulation.
  - This is based on the principle that LASER destroys the capillaries and thus the arterial blood flowing to that region is decreased
- Also, in macular edema, FA allows delineation between ischemia and gravitational edema as a cause of decreased macular function:
  - This is understood by the size of the FAZ
  - If the FAZ is normal, the patients visual function improves with LASER
  - In presence of enlarged FAZ, focal LASER is contraindicated

5. Explain the reason for the development of a cherry-red spot in the eye with central retinal artery occlusion. (3 marks)

- In cases of acute central retinal arterial occlusion, there is global ischemia of the retina leading to cloudy edema of the outer retinal layers. This is the cause for the pale appearance of the retina
- However, the fovea is supplied through the choriocapillaris and thickness is so less (owing to the absence of nerve fiber layer) that the underlying choroidal hue stands out as the characteristic cherry-red spot
- Cherry red spots can also be seen in other conditions
- The cherry-red spot disappears in a span of a few weeks as the edema decreases

6. Describe the fundus findings of central retinal artery occlusion. (3 marks)

- Fundus examination in a central retinal arterial occlusion shows diffuse whitening (due to edema) of the retina with arterial narrowing
- The fovea appears reddish-brown due to the underlying choroid. This phenomenon is called 'cherry red spot'
- The arterioles may show a segmented column of blood due to stagnation of flow. This is called the cattle-trucking phenomenon
- If there is a cilioretinal artery present, the area supplied by it (papillomacular bundle) stands out against the surrounding pale retina and the vision is 6/6 with a severely contracted visual field
- Various emboli may be seen in the arterial arcade
  - Calcific emboli—from calcified valves. they are very dangerous, as they cause permanent obstruction.
  - Fibrin platelet—gray appearing, lead to amaurosis fugax and then complete obstruction. they are usually multiple
  - Cholesterol emboli—Hollenhorst plaques, rarely cause obstruction but appear as golden-colored plaques within the arterioles
- After around 6–8 weeks, cherry red spot and cloudiness disappear, and consecutive optic atrophy develops

7. Contrast Central retinal artery occlusion and ophthalmic artery occlusion. (3 marks)

CRAO and Ophthalmic Artery Occlusion

Feature	Central retinal artery occlusion	Ophthalmic artery occlusion
• Common cause	• Emboli arising from the ipsilateral carotids • Thromboembolic phenomena	• Prolonged compression of the eye, acute blood loss, hypotension, anemia • Vasculitis
• Pathology/site of occlusion	• At the lamina cribrosa	• Behind the lamina cribrosa
• Visual acuity	• Perception of light is positive	• Perception of light negative
• IOP	• Normal	• Low normal
• Fundus examination	• Pale fundus with cherry red spot • Arteriolar attenuation, normal veins	• Pale fundus without cherry-red spot • Attenuated arteries and veins

Contd.

Feature	Central retinal artery occlusion	Ophthalmic artery occlusion
• Fluorescein angiogram	• Choroidal perfusion is normal • Poor perfusion of the arterial tree and delayed arteriovenous circulation time	• Absence of perfusion in the retinal and choroidal circulation around the optic disc • Amalric sign: Triangular areas of nonperfusion seen in the posterior pole

8. Explain various emboli that may be seen in association with retinal artery occlusion. (3 marks)

1. **Calcific emboli**
    - From calcified valves. they are very dangerous, as they cause permanent obstruction
2. **Fibrin platelet**
    - Gray appearing, lead to amaurosis fugax and then complete obstruction. They are usually multiple
3. **Cholesterol emboli**
    - Hollenhorst plaques, rarely cause obstruction but appear as golden-colored plaques within the arterioles

9. Enumerate six rare causes for retinal artery occlusion. (3 marks)

1. Giant cell arteritis
2. Vegetations from bacterial endocarditis
3. Myxomatous cells from myxomas
4. Inflammatory periarteritis as in SLE, PAN, Wegener's granulomatosis, and Behcet's syndrome
5. Thrombophilic disorders
6. Sickle cell anemia

10. Describe the fundus findings of Central retinal artery occlusion 6 weeks after the acute attack. (3 marks)

- The retinal clouding characteristic of CRAO disappears soon
- The cherry-red spot disappears
- The optic disc is pale with a saucer-shaped cup with narrowed arteries.

11. Enlist three extraluminal causes for central retinal artery occlusion. (3 marks)

1. Orbital tumor compressing the distal aspect of the optic nerve
2. A sudden increase in intraocular pressure >60 mm Hg (as in following intravitreal injection)
3. Orbital compartment syndrome

12. Describe the clinical features of branch retinal artery occlusion. (3 marks)

- Branch retinal artery occlusion occurs due to obstruction at the level of bifurcation of the arteries
- Patients may present with central visual loss or peripheral vision loss depending on the arterial arcade affected

- The affected sector of the retina appears pale with attenuated arteries. The occlusion may be due to emboli like calcific, atheromatous, or cholesterol emboli
- However, with time, there is recanalization of the affected vessel and the retina looks normal
- Visual field analysis may show a permanent field defect

13. Describe the appearance of: (1 mark each)

A. Amalric sign
B. Elschnig spots
C. Cattle-trucking of vessels

1. **Amalric sign**
   - Amalric sign is seen in ophthalmic artery occlusion
   - In ophthalmic artery occlusion, triangular areas of retinal whitening seen in various quadrants because of ischemia
   - It is characteristic of posterior ciliary artery occlusion
2. **Elschnig spots**
   - They are a choroidal manifestation of systemic hypertension
   - They appear due to fibrinoid necrosis of the choroidal vessels secondary to an acute rise in systemic blood pressure
   - They are small black spots that are seen in the outer retina with a yellow halo
3. **Cattle-trucking of vessels**
   - It is seen in CRAO
   - The arterioles show a segmented column of blood due to stagnation of flow. This phenomenon is called cattle trucking

14. "A cilioretinal artery occlusion may accompany a central retinal venous occlusion". Is this statement true or false? Justify your answer with reason. (3 marks)

Yes, this statement is true.

Justification

- The cilioretinal artery is seen in a small proportion of individuals (ranging from 6–40% of the population) and its obstruction may be seen in three circumstances
  - Isolated cilioretinal obstruction
  - Associated with central retinal venous occlusion
  - Associated with ischemic optic neuropathy
- The cilioretinal artery arises from the ciliary circulation
  - Either from the peripapillary choroid or one of the short posterior ciliary arteries
  - It is seen arising from the lateral aspect of the disc and runs a short course to supply the macula
- The statement mentioned in the question is true
- The causes for the same are:
  1. A central retinal venous occlusion is usually associated with a raised intraocular pressure. As the cilioretinal artery arises from the ciliary circulation which has a low mean arterial pressure owing to several collaterals, it is easily compressed by increased IOP

2. Also, the choroidal circulation is densely innervated by the sympathetic plexus. It lacks the autoregulation that the retinal circulation has. So, systemic hypertension, which is a risk factor for CRVO too, can affect the perfusion through the cilioretinal artery

15. Contrast Choroidal Circulation and Retinal Circulation. (3 marks)

Choroidal and Retinal Circulation

Feature	Choroidal circulation	Retinal circulation
• Supply	• Supplies the choroid, optic nerve head, Peripapillary retina, outer layers of fovea • Choroid, ciliary body, and a part of the iris	• The retina only
• Appearance of FFA	• Lobular arteriocentric distribution • Fills earlier than retinal circulation	• Arterial tree • Fills after the choroidal circulation
• Autoregulation	• Absent	• Present
• Influence by systemic hypertension	• Highly influenced • Densely innervated by the sympathetic system	• Not influenced • Lacks autonomic regulation
• Presence of collaterals	• Yes, extensive	• No
• Blood-retinal barrier	• Absent due to fenestrated capillaries	• Present
• Watershed zone	• Usually vertical, centered around the disc	• Across the horizontal raphe

16. Describe the clinical features of Precapillary Retinal Artery Occlusion. (3 marks)

Precapillary retinal artery occlusion occurs when there is an acute occlusion of the peripapillary retinal network of vessels.

Symptoms

Patients present with sudden painless visual diminution.

Signs

- Patients present with visual loss and visual field defects depending on the location of the infarct
- There is a nerve fiber layer infarct in the peripapillary area, cotton wool spots, and whitish ischemic areas perpendicular to the optic disc
- They usually disappear after 6 weeks and are peripapillary retinal depressions

17. Describe the clinical features of Ophthalmic Artery Occlusion. (3 marks)

- Ophthalmic artery occlusion occurs in the proximal aspect of the ophthalmic artery well before the ciliary and the CRA arise from it
- Common causes are hypotension, severe anemia, prolonged ocular compression, vasculitides, and severe blood loss (as a part of perioperative visual loss)

Symptoms

The features are sudden painless loss of vision.

Signs

- On examination, there is no perception of light as both the ciliary and the retinal circulation are affected
- The IOP is on the lower side and the pupil demonstrates an afferent pupillary defect

- Fundus shows diffuse pallor of the retina without any cherry-red spot. There is diffuse attenuation of retinal arteries and veins. Amalric sign may be positive
- FFA shows a delayed arm-retina circulation time and poor perfusion of the choroid and the retinal circulation

18. Enumerate the ocular and systemic risk factors associated with retinal venous occlusion. (3 marks)

Ocular and Systemic Risk Factors

Ocular risk factors	Systemic risk factors
• Acute angle-closure glaucoma: A sudden increase in intraocular pressure causes compression of the central retinal vein at the disc • Chronic open-angle glaucoma is a known ocular risk factor for the development of venous occlusion	For CRVO • Arterial hypertension • Diabetes mellitus • Open-angle glaucoma For BRVO • Arterial hypertension • Cardiovascular disease • Increased body mass index • Open-angle glaucoma

19. Explain the arrangement of capillaries at the macula and their importance. (3 marks)

- Retinal capillaries arise from the branching of the arteries. They have a basement membrane with endothelium and a cuff of pericytes
- Sometimes, the macula is supplied directly from the ciliary circulation through the cilioretinal artery
- The capillaries are present in the inner nuclear layer and ganglion cell layer. In the macula, it is a lot denser. There are four layers of capillary plexus
- They are present in the nerve fiber layer, ganglion cell layer, above and below the inner nuclear layer
- This density of capillaries is required to nourish the multiple layers of ganglion cell layers in the macula
- In the central 500 microns, the blood vessels are absent and this is known as the foveal avascular zone. These layers derive nourishment from the choriocapillaris
- In fluorescein angiography, an increase in the size of the FAZ is characteristic of ischemic maculopathy. In these cases, LASER photocoagulation is contraindicated and the visual prognosis remains low

COMPETENCY

OP8.2 ENUMERATE THE INDICATIONS FOR LASER THERAPY IN THE TREATMENT OF RETINAL DISEASES (INCLUDING RETINAL DETACHMENT, RETINAL DEGENERATIONS, DIABETIC RETINOPATHY AND HYPERTENSIVE RETINOPATHY)

SHORT ESSAYS

1. Describe the role of LASERS in ophthalmology. (5 marks)

- The use of LASERs has become an important part of diagnostics and therapeutics in ophthalmology
- LASERs act by three mechanisms:
  1. Thermal effect (photocoagulation, photodisruption, photovaporization)
  2. Photochemical (photoradiation and photoablation)
  3. Ionizing effect

Uses of LASER in Diagnostics

OCT	Uses 810 nm near-infrared laser to produce high-resolution images
Scanning laser ophthalmoscopy	Retinal imaging

Uses of LASER in Therapeutics

Segment	**Wavelength**	**Mechanism and uses**
		Anterior segment
Excimer LASER (photoablation)	193 nm	• Used in refractive surgeries like photorefractive keratectomy, laser *in situ* keratomileusis (LASIK)
Femtosecond LASER	1053 nm	• Used mainly in refractive surgeries to create LASIK flaps • Also, it is being used to create corneal incisions and capsulorhexis in cataract surgeries
Nd: Yag LASER (photodisruption)	1064 nm	• For treating after cataract (most common indication) • For peripheral iridotomy (for angle-closure glaucoma) • Membranectomy (pupillary block glaucoma) • Hyaloidotomy (in malignant glaucoma)
Argon LASER (photocoagulation)	514 nm	• Trabeculoplasty • Cyclophotocoagulation • Argon LASER iridoplasty
		Posterior segment
Argon LASER (photocoagulation)	488–514 nm	Vasculoproliferatve disorders • It acts by destroying hypoxic tissues and thereby reducing the production of VEGF • Examples are proliferative diabetic retinopathy, neovascular glaucoma, Eales' disease Retinal tumors • It acts by cutting off the vascular supply to the tumor • Used in small retinoblastoma and melanomas • Peripheral retinal degenerations and retinal tears • Acts by creating adhesions between the neurosensory retina and pigment epithelium Macular disorders • Extrafoveal choroidal neovascular membrane • Central serous chorioretinopathy (by increasing the pump function of the RPE) • Macular edema (Grid LASER: decreasing the amount of arterial blood reaching the macula, focal LASER: By the destruction of the microaneurysms)
Diode LASER	810 nm	• Panretinal photocoagulation
Infrared LASER	689 nm	• Used in photodynamic therapy for treating choroidal neovascularization and retinal tumors
Nd: Yag LASER	1064 nm	• Pattern scan LASER for diabetic retinopathy (Pascal)

2. Describe the utility of LASER in retinal conditions. (5 marks)

1. **Vasculoproliferative disorders**
    - The principle of LASER photocoagulation is converting hypoxic areas to the anoxic retina so that they do not produce VEGF and thus neovascularization is prevented
    - This principle is used in retinoproliferative disorders
    - Common indications are proliferative diabetic retinopathy, macular edema, neovascular glaucoma due to venous occlusions, retinopathy of prematurity, Eales' disease, Coats' disease, and sickle cell retinopathy
2. **Peripheral retinal degenerations and tears**
    - Here, the principle is to create adhesions between the neurosensory and pigment epithelium thus prevent leakage of subretinal fluid into the subretinal space
    - This prevents the formation of rhegmatogenous retinal detachment
    - A spot size of 200 µm is used to create burns of two concentric circles around the tear
3. **Macular disorders**

Central serous retinopathy	Laser photocoagulation is indicated only if 1. Serous detachment persists >3 months 2. Recurrence of the disease in the eye has visual morbidity due to the same. 3. The permanent visual deficit is present in the fellow eye due to CSCR 4. Occupational requirement 200 µm spot size of laser is applied at the site of leakage found on FFA
Age-related macular degeneration	Indicated in extrafoveal choroidal neovascular membrane

4. **Retinal tumors**

Localized choroidal melanoma and retinoblastoma, with no subretinal seeding or vitreous seeding, are treated with confluent LASER spots around the tumor to cut off the blood supply to the tumor.

3. Describe the indications of LASER in diabetic retinopathy and describe the parameters for LASER. (5 marks)

Laser is indicated in diabetic retinopathy under two conditions:

1. Proliferative retinopathy with high-risk characteristics
2. Macular edema

Proliferative diabetic retinopathy	Parameters of LASER
• Proliferative diabetic retinopathy with high-risk characteristics needs to be treated immediately • High-risk characteristics are: ▪ New vessels at the disc (NVD) > 1/3 disc area ▪ New vessels at the disc (NVD) with preretinal or vitreous hemorrhage ▪ New vessels elsewhere (NVE) >1/2 disc area with preretinal or vitreous hemorrhage	• LASER pan-retinal photocoagulation (ablating the entire surface of retina excluding macula with argon LASER) is the treatment of choice • Blue-green LASER is used, with a spot size of 200–500 µm, 0.1–0.5 s • It is usually completed in 3–4 sessions, quadrant by quadrant • If PDR is associated with macular edema, macular LASER is done before PRP to avoid edema and exudative detachment at the macula

Macular edema	**Parameters of LASER**
Clinically significant macular edema 1. Presence of macular thickening within 500 µm from the foveola 2. Presence of hard exudates within 500 µm from the foveola 3. Presence of hard exudates of 1500 µm size or larger, a part of which is within 500 µm of the foveola	• Argon green, 0.1 s, with a spot size of 50–100 µ • Focal LASER (ablation of microaneurysms) by applying LASER burns of size 50–100 µ mover microaneurysms and centers of rings of hard exudates • The principle is to destroy microaneurysms and thus prevent leakage • Grid LASER is applied 500 µm outside the fovea within the arterial arcade, of spot size 100 µm
FFA is used to differentiate focal, diffuse, and ischemic maculopathy	LASER is contraindicated in ischemic maculopathy

4. Describe the indications, parameters, and mechanism of LASERs in macular disorders. (5 marks)

Indications, Parameters, and mechanism of LASER

Indication	**Parameters**	**Mechanism of laser**
1. ***Diabetic macular edema*** Clinically significant macular edema i. Presence of macular thickening within 500 µm from the foveola ii. Presence of hard exudates within 500 µm from the foveola iii. Presence of hard exudates of 1500 µm size or larger, a part of which is within 500 µm of the foveola FFA is used to differentiate focal, diffuse, and ischemic maculopathy	• Argon green, 0.1s, with a spot size of 50–100 µ • Focal LASER (ablation of microaneurysms) by applying LASER burns of size 50–100 µm over microaneurysms and centers of rings of hard exudates • Grid LASER is applied 500 µm outside the fovea within the arterial arcade, of spot size 100 µm	• The focal laser acts by destroying the microaneurysms that are responsible for exudation • Grid laser acts by reducing the leaking capillaries bringing arterial blood to the macula
2. ***Macular edema in venous occlusions*** It is confirmed by FFA and OCT. LASER is not given if FAZ >500 µm in size.	• Grid LASER is applied 500 µm outside the fovea within the arterial arcade, of spot size 100 µm	• Grid laser acts by reducing the leaking capillaries bringing arterial blood to the macula
3. **Central serous chorioretinopathy** • Serous detachment persists >3 months • Recurrence of the disease in the eye which has visual morbidity due to the same • A permanent visual deficit is present in the fellow eye due to CSCR • Occupational requirement	• Focal argon green laser (parameters like the focal laser) or, • Subthreshold micropulse LASER photocoagulation • Yellow dye laser 577 nm	• It acts by increasing the RPE pump function

Contd.

Indication	Parameters	Mechanism of laser
4. **Age-related macular degeneration** • Classic choroidal neovascular membrane with definite boundaries • Extrafoveal or juxtafoveal. Contraindicated in subfoveal CNVM	• Argon green LASER • Frequency-doubled YAG laser	• The laser helps to seal the leaking blood vessels that give rise to exudative detachment

SHORT ANSWERS

1. Explain the principles of using LASER and transpupillary thermotherapy in retinal tumors. (3 marks)

- In retinal tumors like retinoblastoma and choroidal melanomas, the laser is used to create burns around the tumor
- 1 or 2 concentric circles of laser burns are created with a spot size of 200–500 μm and 0.5–1 s duration
- This destroys blood vessels in the choroid that supply the tumor. As a result, the tumor is destroyed by a loss of blood supply
- Thermotherapy is performed by using ultrasound, microwave, or infrared radiation to create heat and cause tumor cell death by heat

2. Explain the LASER settings used in the treatment of diabetic maculopathy. (3 marks)

Laser Settings for Macular edema

- Argon green, 0.1 s, with a spot size of 50–100 μ
- Focal LASER (ablation of microaneurysms) by applying LASER burns of size 50–100 μm over microaneurysms and centers of rings of hard exudates
- The principle is to destroy microaneurysms and thus prevent leakage
- Grid LASER is applied 500 μm outside the fovea within the arterial arcade, of spot size 100 μm
- LASER is contraindicated in ischemic maculopathy

3. Enumerate the indications of LASER in ROP. (3 marks)

Retinopathy of prematurity is a vasculoproliferative disorder due to VEGF secreted from the avascular retina. Due to the VEGF produced, there is a production of new vessels that cause tractional retinal detachment and blindness.

Indications

1. **Threshold ROP**
    - The threshold is a state at which intervention is required
    - It is defined as stage 3+ in the zone I or II involving 5 contiguous clock hours or 8 noncontiguous clock hours
    - The peripheral avascular retina is ablated with Argon laser
2. **Aggressive posterior ROP or rush disease**
    - ROP in the zone I or II with features of the plus disease (dilatation of vessels in the posterior pole at least in 2 quadrants)

3. **Type I prethreshold ROP**
   - Zone I ROP, any stage with plus
   - Zone I ROP, stage 3, non-plus
   - Zone II ROP, stage 2 or 3, with plus disease

4. Describe the indications of LASER in peripheral retinal degenerations. (3 marks)

- Here, the principle is to create adhesions between the neurosensory and pigment epithelium thus prevent leakage of synchitic fluid into the subretinal space
- This prevents the formation of rhegmatogenous retinal detachment
- A spot size of 200 µm with a duration of 0.1–0.2 s is used to create burns of two concentric circles around the tear to create moderate retinal whitening
- Thus, for prophylaxis, it is helpful in
  - Lattice degeneration, areas of white with pressure and white without pressure are areas of increased vitreous adhesion and may lead to the formation of tears
  - Retinal tears need to be treated, especially if they are large, in the superior quadrant and if they are symptomatic (associated with photopsia)

COMPETENCY

OP8.3 DEMONSTRATE THE CORRECT TECHNIQUE OF A FUNDUS EXAMINATION AND DESCRIBE AND DISTINGUISH THE FUNDUSCOPIC FEATURES IN A NORMAL CONDITION AND IN CONDITIONS CAUSING AN ABNORMAL RETINAL EXAMINATION

SHORT ESSAYS

1. Explain the parts of a normal retina when seen using an ophthalmoscope. (5 marks)

Parts of Normal Retina (Fig. 8.3.1)

1. **Media**
   - Media includes the cornea, anterior chamber, lens, and vitreous
   - It should be clear, and a red glow should be seen on distant direct ophthalmoscopy
2. **Optic disc**
   - The disc is seen on the nasal side a vertical oval vermillion colored, around 1500 microns in size
   - It has a central depression called the cup, from which four major sets of vessels are seen to arise

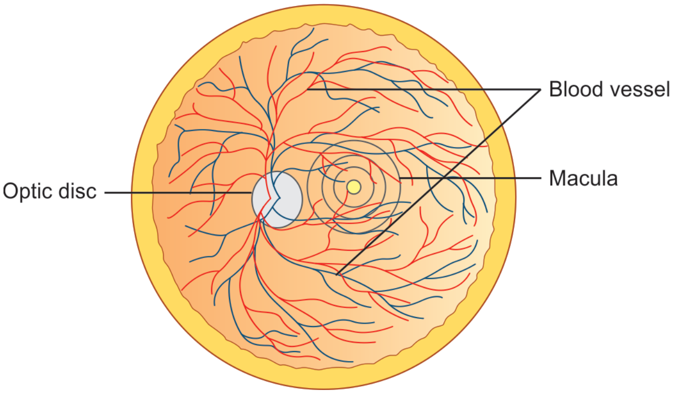
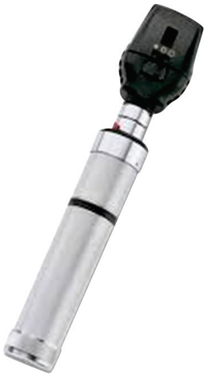


Fig. 8.3.1: Anatomy of the retina

- Shape, size, color, and margins are noted
- The normal shape of the disc is round or vertical oval. Color is vermillion and the cup appears as a pale depression. Margins are distinct

3. **Vessels**
   - The vessels arise from the center of the cup and are transparent
   - The blood column is seen as a continuous column
   - The thickness of arteries and veins is of 2:3 ratio
4. **Macula**
   - The macula is the area between the superior and inferior temporal arcades
   - In its center is a shining pit called foveola (0.35 mm diameter) which is situated about 2 disc-diameters (3 mm) away from the temporal margin of the disc and about 1 mm below the horizontal meridian
   - An area of about 500 μm in diameter does not contain any retinal capillaries and is called the foveal avascular zone (FAZ)
   - Surrounding the fovea are the parafoveal and perifoveal areas

2. Identify the instrument. Explain the procedure. (1+4 marks)



The Instrument Shown is

- Direct ophthalmoscope
- Used to view the retina

Procedure of Funduscopy

- Light is shone into the patient's eyes from a distance of 25 cm to observe the red glow. This is called distant direct ophthalmoscopy. It helps to pick up any opacities in the media of the eye like corneal opacities, cataracts, and vitreous opacities
- Depending on the uncorrected refractive error of the patient and the examiner, the dial in the ophthalmoscope is corrected
- Then, standing on the temporal aspect of the patient, the observer moves close to the eye to focus on the retina while instructing the patient to look at a distant object

Viewing the Optic Disc

- The most prominent structure seen on the nasal aspect of the retina is the optic disc
- The right eye of the patient is observed with the right eye of the observed, and the left eye of the patient is observed with the left eye
- The size, shape, color, and margins of the disc are noted
- The position and size of the cup are noted
- The vessels are seen to arise from the center of the disc

Viewing the Vessels

- The vessels are followed till the view allows
- Their thickness and course are noted

Viewing the Macula

- This is followed by the examination of the macula or the area centralis
- A central bright reflex is seen in the fovea known as the foveolar reflex

Viewing the Peripheral Retina

- The periphery of the retina is not visible by direct ophthalmoscopy
- Nonetheless, the retina can be examined till the equator and slightly beyond

3. Compare and contrast a direct ophthalmoscope with an indirect ophthalmoscope. (5 marks)

Direct and Indirect ophthalmoscope

Feature	Direct ophthalmoscope	Indirect ophthalmoscope
Field of view	Smaller	Large
Need of a condensing lens	No	Yes
Effect of refractive error on image	Needs adjustment for refractive error	Not necessary
Characteristics of image	The image formed is erect and thus no confusion regarding lateral inversion	The image formed is real and inverted (laterally and vertically)
Size of the image	1.5 times magnified	1.5 times magnified
Binocularity	Absent	Present
Stereopsis	Absent	Present
Measuring depth/elevation	Possible	Not possible
Working distance	Small (1–2 cm), may make the patient uncomfortable	More than in direct ophthalmoscopy (20–25 cm)
Structures best seen	Optic disc Macula	Retinal periphery
Mastering the skill	Easy	Difficult
View in hazy media	Difficult	Easy

SHORT ANSWERS

1. Describe the characteristics of an image formed by a direct ophthalmoscope. (3 marks)

- The image formed by a direct ophthalmoscope is a virtual image formed behind the retina of the patient (Fig. 8.3.2)

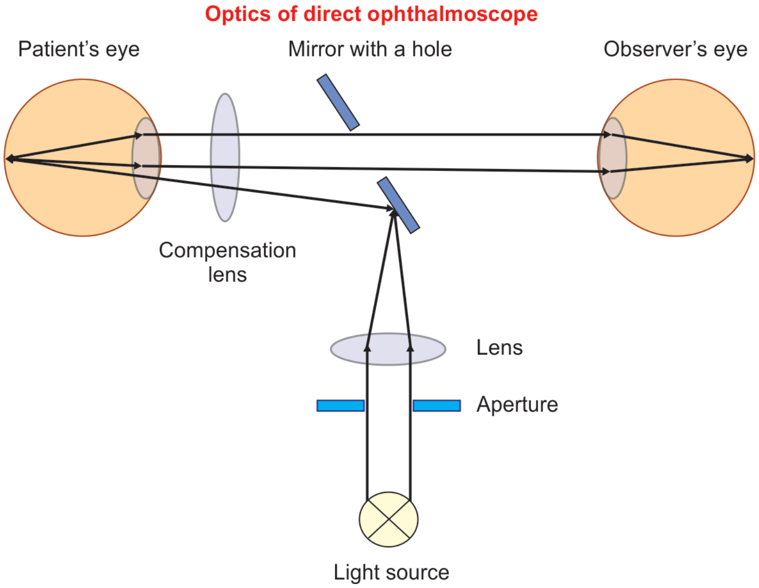


Fig. 8.3.2: Image formation by direct ophthalmoscope

- It is erect and magnified up to 15 times
- The field of vision is low
- In the direct ophthalmoscope elevation and depression of structures (e.g., disc edema, cup excavation) can be measured in diopters using the dial

2. Define area centralis and describe its parts. (3 marks)

Area centralis is the area between the superotemporal and inferotemporal vascular arcades. It contains the macula (Fig 8.3.3).

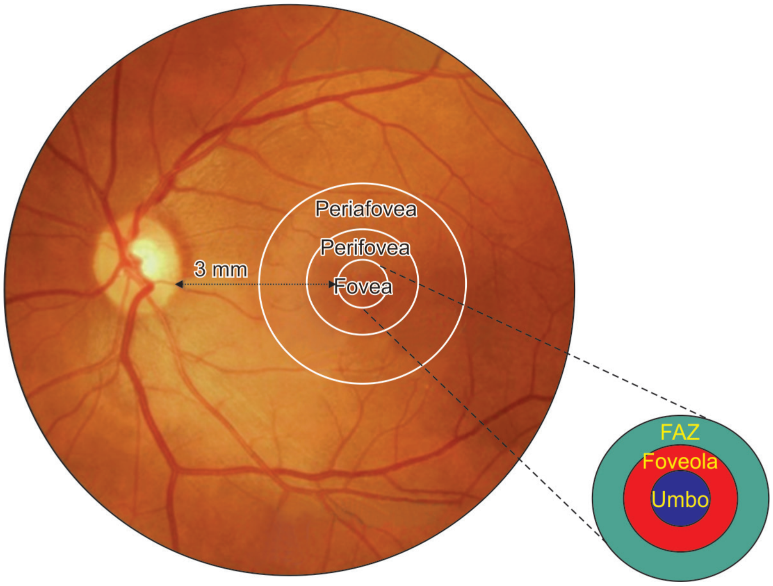


Fig. 8.3.3: Area centralis

Parts

1. **Macula**
   - The macula is 5.5 mm in diameter and is yellowish due to the presence of xanthophylls
2. **Fovea centralis**
   - Fovea centralis the central depression and about 1.85 mm in diameter
   - It corresponds to the central 5° of the visual field
3. **Foveola**
   - In its center is a shining pit called foveola (0.35 mm diameter) which is situated about 2 disc diameters (3 mm) away from the temporal margin of the disc and about 1 mm below the horizontal meridian
   - It contains a shining pit called the umbo which corresponds to the foveal reflex seen clinically
   - An area of about 500 µm in diameter does not contain any retinal capillaries and is called the foveal avascular zone (FAZ). It is present within the fovea but outside the foveola
   - Surrounding the fovea are the parafoveal (0.5 mm) and perifoveal areas (1.5 mm)

3. Describe the causes for abnormalities of media and explain how they are differentiated. (3 marks)

- Medial haze can be due to corneal, anterior chamber, lens, and vitreous opacities
- It is best appreciated on distant direct ophthalmoscopy
- If the opacity moves in the same direction as the movement of the eye, then the opacity is anterior to the nodal point (posterior capsule of lens). If it moves in the opposite direction, then it is posterior to the nodal point
- Common causes for the former are corneal opacity, hyphema, and cataracts
- Causes for the latter include vitreous hemorrhage and vitreous opacities

4. Describe the appearance of (1 mark each)

A. Flame-shaped hemorrhages
B. Hard exudates
C. Cotton wool spots

A. Flame-shaped Hemorrhages

These are superficial retinal hemorrhages with varying shapes and feathery margins.

B. Hard Exudates

- These are intraretinal yellow, well-demarcated lesions, usually seen in the posterior pole around a leaking microaneurysm or capillary
- They are lipid deposits in the deep layers of the retina where the axons are tightly packed perpendicular to the surface of the retina

C. Cotton Wool Spots

- They are yellow-white, cotton-like fluffy appearing, superficial lesions with fuzzy borders, and are present in the nerve fiber layer
- They are usually seen in association with acute ischemia

5. Describe the normal periphery of the retina. (3 marks)

The peripheral retina is divided into four regions (Fig. 8.3.4)

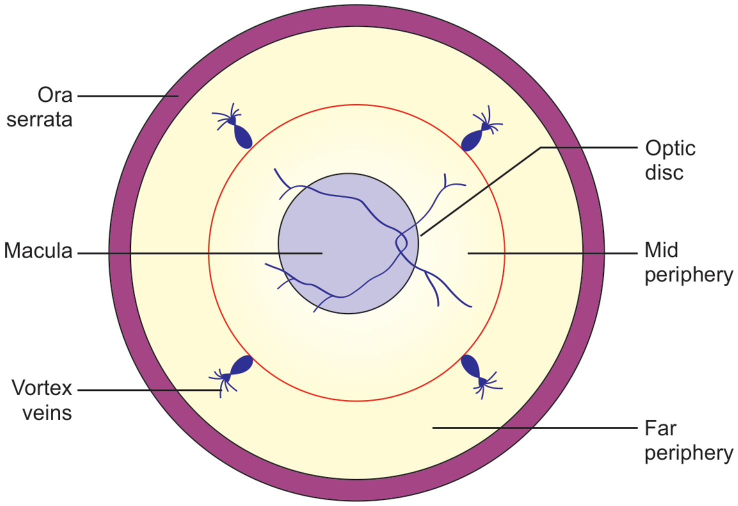


Fig. 8.3.4: Parts of peripheral retina

1. **Near periphery**
    - 1.5 mm region around the area centralis
2. **Mid periphery**
    - 3 mm zone around the near periphery
3. **Far periphery**
    - 9–10 mm on the nasal side from the optic disc and 16 mm on the temporal side from the optic disc
4. **Ora serrata**
    - Serrated peripheral margin where the retina ends and the ciliary body starts
    - At the ora serrata, the neurosensory retina and RPE adhere to each other and the vitreous base is tightly adherent
    - It is 6 mm from the nasal limbus and 7 mm from the temporal limbus

6. Describe abnormalities of the optic disc that can be detected on a routine fundus examination. (3 marks)

Abnormalities can be grouped under color, shape, size, and margins for examination

Property	Normal	Abnormal
Color	It is vermillion in color	The pallor of the disc • is the hallmark of optic atrophy • It can be diffuse pallor (as in primary optic atrophy), sectoral (as in AION), and bowtie (pituitary tumors) Hyperemia • It can be seen in inflammation and papilledema
Shape	The disc is usually round or vertically oval	• Oblique discs are seen in high astigmatism and tilted disc syndrome
Size	It is normally 1500 μm	• Small discs are seen in hypermetropes, hypoplastic discs, and colobomata • Large discs are seen in myopes, morning glory syndrome and megalopapilla
Margins	They are sharp and distinct normally	• Blurred margins are seen in disc edema and congenital anomalies of disc • Elevation of the disc surface is pathognomonic of edema
Vessels	Venous pulsations can be seen in around 80% of the normal population and can be elicited by digital pressure on the globe in the rest	• It is absent in increased intracranial tension leading to papilledema • It is also absent in elevated intraocular pressure

7. Explain the normal appearance of blood vessels in the retina. (3 marks)

- The blood vessels of the retina appear to arise from the center of the cup
- There are four branches
    1. Superotemporal
    2. Inferotemporal
    3. Superonasal
    4. Inferonasal vascular arcade
- The temporal vessels are arched towards the macula and the nasal vessels are radiating towards the periphery

- The arteries and veins always travel together and have a common adventitious sheath. They may cross each other through their course
- The diameter of the vessels decreases away from the center. The ratio of the caliber of the vessels is 2:3 (artery: vein)
- The vessel walls are so thin to allow the visualization of the inner blood column. The arteries appear bright red and the veins appear dull red

8. Justify the brownish appearance of the macula. Explain how the histology of the macula is different from the rest of the retina. (3 marks)

- The brownish appearance of the macula is due to the tall RPE cells that are pigmented
- Also, the xanthophyll pigments give a yellowish hue

Histology of Macula vis-à-vis Rest of the Retina

1. Relatively a greater number of cones
2. One to one correspondence between photoreceptor and bipolar cells
3. Longer cones
4. Taller RPE with increased pigmentation
5. Presence of xanthophyll (very sensitive to blue light)—basis for macular function tests
6. Two or more layers of ganglion cells layer, midget ganglion cells in abundance
7. Obliquely arranged outer plexiform layer
8. Four layers of capillary network vis-à-vis two layers in the rest of the retina

9. Enumerate the three layers of the fovea. (3 marks)

1. Retinal pigment epithelium
2. Layer of cones
3. Internal limiting membrane

10. Describe the appearance of (1 mark each)

A. Lattice degeneration
B. White with pressure
C. Snail tract degeneration

A. Lattice Degeneration

- These are usually bilateral, spindle-shaped areas of retinal thinning located between the equator and the vitreous base
- It consists of sclerosed vessels with an arborizing network of white lines

B. White with Pressure

- Retinal areas with a translucent white-gray appearance that are induced by scleral indentation
- They are seen in normal eyes where the vitreous is tightly adherent

C. Snail Tract Degeneration

Sharply demarcated bands of tightly packed snowflake-like appearance that give the retina a white frost-like appearance.

11. Describe variations in the appearance of blood vessels of the retina. (3 marks)

Variations in the Appearance of Blood Vessels

- Arteries are attenuated in hypertension, vascular occlusions, and post-inflammatory states
- Arterial tortuosity is rare and is seen in the plus disease of ROP
- In arterial occlusions, the arterioles may show a segmented column of blood due to stagnation of flow. This is called the cattle trucking phenomenon
- In certain types of retinal vasculitis, there is sheathing of the arteries and veins
  - The sheathing can be patchy or continuous
  - It is called Kyrieleis arteritis when associated with infectious retinitis and is seen as segmental sheathing
  - Arterial sheathing with hemorrhages is characteristically seen in association with acute retinal necrosis
- Carotid emboli
  - Usually arise at the bifurcation, and since the left internal carotid has a straighter course, left CRAO is more common due to embolic phenomena
  - Calcific emboli—from calcified valves. they are very dangerous, as they cause permanent obstruction
  - Fibrin platelet—gray appearing, lead to amaurosis fugax and then complete obstruction. they are usually multiple
  - Cholesterol emboli—Hollenhorst plaques, rarely cause obstruction but appear as golden-colored plaques within the arterioles

Variations in Appearance of Veins

- Venous dilatation is seen in hypercoagulable states, diabetic retinopathy, and partial obstruction of veins. It is also seen in the plus disease of ROP
- Collaterals can be seen as minute vessels across the horizontal raphe
- Venous sheathing is seen in sarcoidosis, Eales'disease, and Behcet's disease

Variations in Capillaries

- New vessels can be seen as fine vessels extending anteriorly
- IRMA or intraretinal microvascular abnormalities are seen as fine shunts between arterioles and venules

12. Give three causes for flame-shaped hemorrhages (3 marks)

1. Retinal venous occlusion
2. Hypertensive retinopathy
3. Anemic retinopathy

13. Differentiate between preretinal and subretinal hemorrhage. (3 marks)

Feature	Preretinal hemorrhage	Subhyaloid hemorrhage
Location	Hemorrhage between the nerve fiber layer and the internal limiting membrane	Hemorrhage between the internal limiting membrane and the posterior limiting layer of the vitreous
The shape of the hemorrhage	Can be of any shape	Usually boat-shaped

14. Describe the appearance of Roth spots. Give two causes. (2+1 marks)

Appearance of Roth Spots

- Roth spots are white-centered intraretinal hemorrhages
- The white center is formed by fibrin thrombus, platelet aggregates, and infiltrating red cells

Causes

1. Bacterial endocarditis
2. Leukemia

15. Explain the appearance of cotton wool spots and enumerate two causes for the same. (2+1 marks)

Appearance of Cotton Wool Spots

They are yellow-white, cotton-like fluffy appearing, superficial lesions with fuzzy borders, and are present in the nerve fiber layer.

Causes

1. Hypertensive retinopathy
2. Diabetic retinopathy

16. Enumerate three conditions associated with optic disc pallor. 3 marks)

1. Ischemic optic neuropathy
2. Previous optic neuritis
3. Chiasmal compression

COMPETENCY

OP8.4 ENUMERATE AND DISCUSS TREATMENT MODALITIES IN MANAGEMENT OF DISEASES OF THE RETINA

LONG ESSAYS

1. A newly diagnosed diabetic has been referred to you by his treating physician.

A. Define diabetic retinopathy (1 mark)
B. Describe the stages of diabetic retinopathy (4 mark)
C. Explain the necessity for screening for diabetic retinopathy (2 marks)
D. Explain the treatment principles for the same (3 marks)

A. Definition of Diabetic Retinopathy

A microvascular angiopathy associated with diabetes mellitus (both types) affecting retinal arterioles, capillaries, and venules, leading to a cascade of vascular occlusion, ischemia, edema, and neovascularization; and in turn, leading to visual loss.

B. Stages of Diabetic Retinopathy

Nonproliferative diabetic retinopathy (NPDR) or background retinopathy	Proliferative diabetic retinopathy (PDR)
• Mild NPDR • Moderate NPDR • Severe NPDR • Very severe NPDR	• Without high-risk characteristics • With high-risk characteristics

Contd.

Nonproliferative diabetic retinopathy (NPDR) or background retinopathy	Proliferative diabetic retinopathy (PDR)
Diabetic maculopathy • Focal maculopathy • Diffuse maculopathy • Ischemic maculopathy	• Advanced diabetic eye disease

Definitions of Each Type of Retinopathy

1. Mild NPDR: The presence of one microaneurysm/ dot/ blot hemorrhage in one quadrant
2. Moderate NPDR: The presence of hemorrhages in 1–3 quadrants
3. Severe NPDR: One of the following 4-2-1 rules
   i. Presence of severe hemorrhages in all four quadrants
   ii. Presence of venous anomalies like looping/beading in 2 quadrants
   iii. Presence of intraretinal microvascular abnormalities (IRMA) in 1 quadrant
4. Very severe NPDR: Two of the following 4-2-1 rules
   i. Presence of severe hemorrhages in all four quadrants
   ii. Presence of venous anomalies like looping/beading in 2 quadrants
   iii. Presence of intraretinal microvascular abnormalities (IRMA) in 1 quadrant
5. Clinically significant macular edema
   i. Presence of macular thickening within 500 μm from the foveola
   ii. Presence of hard exudates within 500 μm from the foveola
   iii. Presence of hard exudates of 1500 μm size or larger, a part of which is within 500 μm of foveola
6. Proliferative diabetic retinopathy: It is classified as with and without high-risk characteristics
   i. New vessels at the disc (NVD) >1/3 of the disc area
   ii. New vessels at the disc (NVD) with preretinal or vitreous hemorrhage
   iii. New vessels elsewhere (NVE) >1/2 of disc area with preretinal or vitreous hemorrhage
7. Advanced diabetic eye disease: It occurs because of uncontrolled PDR. Its features are tractional retinal detachment, persistent vitreous hemorrhage, and neovascular glaucoma

C. Necessity to Screen

- Diabetic retinopathy is asymptomatic in the initial stages
- The only stages where diabetic retinopathy is symptomatic are diabetic macular edema and complicated proliferative diabetic retinopathy (by vitreous hemorrhage and tractional detachment)
- These are late stages, and the visual prognosis is always guarded
- When screened regularly following dilatation of the pupil, the early nonproliferative stages can be picked up by the ophthalmologist
- The screening protocol is as follows:

Mild NPDR	Annual review
Moderate NPDR	Review every 4–6 months
Severe NPDR	Review every 3 months
Very severe NPDR	Review every 2 months

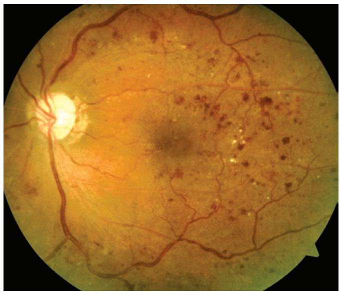
D. Principles for Treatment

1. Nonproliferative diabetic retinopathy needs to be screened in the following manner
   - The strict control of blood sugars and associated risk factors (hypertension, hyperlipidemia, anemia, renal parameters) after pregnancy, diabetic retinopathy tends to recede, hence there is no need for urgent treatment

Mild NPDR	Annual
Moderate NPDR	Every 4–6 months
Severe NPDR	Every 3 months
Very severe NPDR	Every 2 months

2. Proliferative diabetic retinopathy with high-risk characteristics needs to be treated immediately
   - LASER panretinal photocoagulation (ablating the entire surface of the retina excluding macula with argon LASER) is the treatment of choice
   - Its principle is the conversion of the hypoxic retina to an anoxic retina so that no more VEGF is released, and hence neovascularization is halted
   - Blue-green LASER is used, with a spot size of 200–500 µm. It is usually completed in 3–4 sessions, quadrant by quadrant
     - If PDR is associated with macular edema, macular LASER is done before PRP to avoid edema and exudative detachment at the macula
   - Another new and promising modality is injecting anti-VEGF agents into the vitreous
     - They are known to shrink the new vessels and also have a role in decreasing edema, e.g. bevacizumab, ranibizumab, aflibercept
     - They are contraindicated in the presence of traction bands
3. Advanced diabetic eye disease needs intraocular surgery in the form of vitrectomy, the release of traction bands, application of tamponading agents to treat retinal detachment
   - In the same setting endo-LASER (application of LASER burns from within) can be done
4. Clinically significant macular edema has the following treatment options:
   - Intravitreal injection of steroid (triamcinolone) or fluocinolone implant
   - Intravitreal injection of anti-VEGF agents
   - Focal LASER (ablation of microaneurysms) by applying LASER burns of size 50–100 µm over the microaneurysms and centers of rings of hard exudates
   - Frequency-doubled Nd YAG LASER: A predetermined array of LASER burns is applied in the form of pattern scan LASER (PASCAL). It is time-saving and more comfortable for the patient
   - Pars plana vitrectomy may be necessary if macular edema is caused by tangential traction from a thickened and taut posterior hyaloid

2. A long-standing diabetic was examined for retinopathy and the following picture is seen.



A. Identify three abnormal lesions associated with diabetic retinopathy that are seen in this image. (3 marks)

B. Which grade of retinopathy is this? (1 mark)

C. How is this patient treated? (1 mark)

D. Explain the grades of diabetic retinopathy and the screening protocol. (5 marks)

A. Three Abnormal Lesions Associated with Diabetic Retinopathy Seen in this Case

1. Blot hemorrhages
2. Hard exudates
3. Venous dilatation

B. Grade of Retinopathy

- The image is of very severe nonproliferative diabetic retinopathy as there are multiple hemorrhages in all the quadrants and there is an IRMA in the inferonasal quadrant
- There is also venous beading in the inferotemporal quadrant

C. Treatment

- This patient needs to be closely monitored for the development of new vessels by screening every 2 months
- Blood sugar levels should be tightly controlled
- Fluorescein angiography should be performed to rule out the presence of new vessels and macular edema
- Optical coherence tomography of the macula also should be performed to rule out macular edema

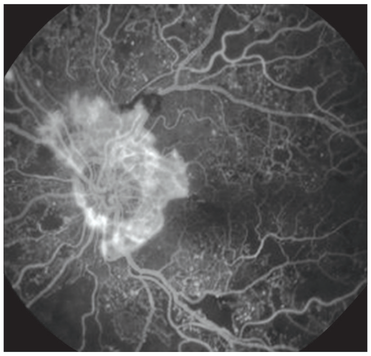
D. Stages of Diabetic Retinopathy and Screening Protocol

Stages	Screening protocol
Mild NPDR: The presence of one microaneurysm/ dot/blot hemorrhage in one quadrant	Annual review
Moderate NPDR: The presence of hemorrhages in 1–3 quadrants	Every 4–6 months
Severe NPDR: One of the following 4-2-1 rules 1. Presence of severe hemorrhages in all four quadrants 2. Presence of venous anomalies like looping/beading in 2 quadrants 3. Presence of intraretinal microvascular abnormalities (IRMA) in 1 quadrant	Every 3 months
Very severe NPDR: Two of the following 4-2-1 rules 1. Presence of severe hemorrhages in all four quadrants 2. Presence of venous anomalies like looping/ beading in 2 quadrants 3. Presence of intraretinal microvascular abnormalities (IRMA) in 1 quadrant	Every 2 months
Clinically significant macular edema 1. Presence of macular thickening within 500 µm from the foveola 2. Presence of hard exudates within 500 µm from the foveola 3. Presence of hard exudates of 1500 µm size or larger, a part of which is within 500 µm of foveola.	Needs treatment in the form of LASER or intravitreal Triamcinolone/anti-VEGF agents
Proliferative diabetic retinopathy It is classified as with and without high-risk characteristics 1. New vessels at the disc (NVD) > 1/3 of the disc area. 2. New vessels at the disc (NVD) with preretinal or vitreous hemorrhage. 3. New vessels elsewhere (NVE) >1/2 of disc area with preretinal or vitreous hemorrhage	Panretinal photocoagulation with Argon LASER Intravitreal anti-VEGF injections
Advanced diabetic eye disease • It occurs because of uncontrolled PDR. • Its features are tractional retinal detachment, persistent vitreous hemorrhage, and neovascular glaucoma.	Vitrectomy

3. A 48-year-old man has been advised an investigation as he was diabetic.

A. Identify the test and comment on one prominent finding. (2 marks)
B. Name two indications for this test. (2 marks)
C. Describe the pathophysiology of diabetic retinopathy. (4 marks)
D. Explain the role of anti-VEGF agents in diabetic retinopathy. (4 marks)

A. Identification

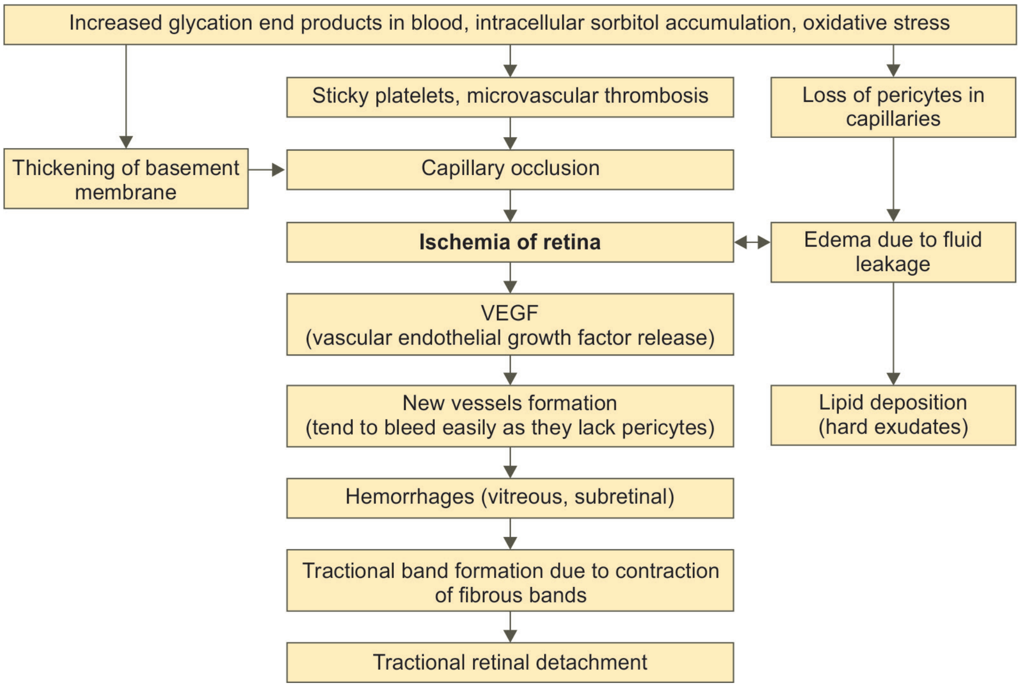


- The investigations shown is fundus fluorescein angiography (FFA)
- A prominent finding seen here is neovascularization at the optic disc

B. Indications for FFA

- To look for new vessels in the retina
- To find the source of a leak in central serous chorioretinopathy

C. Pathophysiology of Diabetic Retinopathy


Increased glycation end products in blood, intracellular sorbitol accumulation, oxidative stress
Sticky platelets, microvascular thrombosis
Loss of pericytes in capillaries
Thickening of basement membrane
Capillary occlusion
Ischemia of retina
Edema due to fluid leakage
VEGF (vascular endothelial growth factor release)
New vessels formation (tend to bleed easily as they lack pericytes)
Lipid deposition (hard exudates)
Hemorrhages (vitreous, subretinal)
Tractional band formation due to contraction of fibrous bands
Tractional retinal detachment

D. Role of Anti-VEGF Agents In Diabetic Retinopathy

Role of VEGF in Causing Retinopathy

- Vascular endothelial growth factor has come to be known as a central molecule in various vasculoproliferative disorders
- It is a subfamily of molecules that are involved as signaling proteins for angiogenesis and vasculogenesis
- VEGF is secreted by the retinal pigment epithelium, pericytes, astrocytes, Muller cells, and endothelial cells

- The ischemia that is induced by capillary occlusion and edema in diabetic retinopathy predisposes to the secretion of VEGF. This molecule causes neovascularization or angiogenesis

Anti-VEGF agents

- The use of anti-VEGF agents is on the rise and is becoming standard of care in proliferative diabetic retinopathy and macular edema
- The drugs available are:
  1. Anti- VEGF antibodies
     i. Bevacizumab
     ii. Ranibizumab
  2. Inhibitors of intracellular transcription
     i. Bevasirnab
  3. Inhibitors of extracellular VEGF
     i. Pegaptanib sodium
  4. Inhibitors of VEGF receptor expression
     i. Aflibercept
  5. Inhibitors of intracellular signaling cascade activating VEGF
     i. Midostaurin
  6. Protein kinase C inhibitor
     i. Ruboxistaurin
- Of these drugs, bevacizumab, ranibizumab, pegaptanib, and aflibercept are commonly used
- The mode of use is intravitreal injection
- These drugs cause a decrease in the new vessel growth and edema. So, their use in proliferative diabetic retinopathy and macular edema is justified
- Though the standard of care for PDR is panretinal photocoagulation (PRP), anti-VEGF injections help to decrease the number of new vessels and this can be followed by PRP
- They are especially of help in cystoid form of macular edema, ischemic maculopathy

Contraindications for the use of Anti-VEGF Agents

1. Presence of traction bands
2. Intraocular inflammation
3. A recent event of an adverse cerebrovascular accident like stroke.

Disadvantages/Adverse Effects of Anti-VEG-F Agents

1. Postoperative endophthalmitis
2. Glaucoma
3. Needs a repeat treatment after 3–4 months

4. A 29-week-gestation premature child is in NICU for prematurity. Her weight is 1200 g.

A. State the retinal condition that may be anticipated in this child. (1 mark)
B. Explain normal vasculogenesis of the retina. (3 marks)
C. How is this condition caused? Explain its pathophysiology. (3 marks)
D. Explain the screening protocol for this condition. (3 marks)

A. Retinal Condition That may be Anticipated in this Child

Retinopathy of prematurity

B. Normal Vasculogenesis of Retina

- Vasculogenesis is the *de novo* formation of new vessels from the precursor cells
- The retina is avascular till about 16 weeks of gestation. Normally, vessels begin to form from the optic disc and develop towards the periphery in a controlled manner under the influence of various growth factors and low oxygen tension
- A group of mesenchymal cells initially grow outwards in the nerve fiber layer from the region of the optic disc to give rise to the capillary system. Some enlarge to form arterioles and venules
- The vessels reach the nasal ora serrata by 36 weeks and temporal ora serrata by 40 weeks of gestation. When this plexus reaches the periphery, it sends vertical offshoots that reach the outer retina
- Astrocytes that are present at the leading edge of the vascular extension produce Vascular endothelial growth factor (VEGF) and control the extracellular matrix. It is complemented by insulin-like growth factor I secretion

C. Pathophysiology of Retinopathy of Prematurity

- When the baby is born prematurely and is associated with some risk factors, there is an increase in oxygen content in the arteries vis-à-vis in the umbilical vein which has lower oxygen content. Also, there is a sudden drop in the IGF-1 and VEGF levels
- It was commonly seen in neonates receiving hyperbaric oxygen after birth and in presence of other risk factors like low birth weight, genetic predisposition, and a short gestational period
- As a result, the vascluogenesis halts
- The area of the retina that is not yet vascularized starts becoming ischemic and secretes VEGF- vascular endothelial growth factor (Michelson's factor X) and insulin-like growth factor I
- This leads to angiogenesis (abnormal new vessel formation). These new vessels are fragile and tend to bleed and then cause tractional RD (Fig. 8.4.1)
- It is seen in as many as 80% of neonates below the gestational age of 28 weeks and birthweight <1500 g

D. Screening Protocol

Timing of Screening

- Gestational age <32 weeks
- Birth weight <1500 g
- The first examination should be done between 6 and 7 weeks postnatal age or 34 weeks post-conceptual age (whichever is earlier)

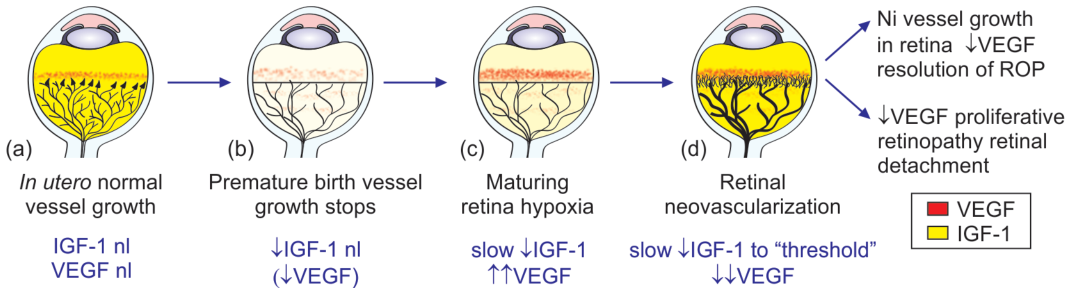

Ni vessel growth in retina ↓VEGF resolution of ROP
↓VEGF proliferative retinopathy retinal detachment
(a)
In utero normal vessel growth
IGF-1 nl
VEGF nl
(b)
Premature birth vessel growth stops
↓IGF-1 nl
(↓VEGF)
(c)
Maturing retina hypoxia
slow ↓IGF-1
↑↑VEGF
(d)
Retinal neovascularization
slow ↓IGF-1 to "threshold"
↓↓VEGF
🟥 VEGF
🟨 IGF-1

Fig. 8.4.1: Pathogenesis of ROP

- Depending on the progression, review screening is performed till the threshold is reached and treatment is given. If ROP does not develop, screening is done till 40 weeks of gestation

Procedure of Screening

- The pupils are dilated with tropicamide 0.5% and phenylephrine 1% (diluted drops)
- The entire retina is screened by indirect ophthalmoscopy preferably with a 30D lens
- Scleral indentation is done to view the extreme periphery

5. An 88-year-old Caucasian male presents with slow onset metamorphopsia noticed from 3 months. On examination, there are golden yellow refractile bodies seen at the macula.

A. State the most probable diagnosis. (1 mark)

B. Explain the pathophysiology of this condition. (3 marks)

C. Describe the fundus appearance of each stage of this condition. (6 marks)

A. Most Probable Diagnosis

Dry age-related macular degeneration

B. Pathophysiology of ARMD

- ARMD has a multifactorial etiology and has two components: Genetic predisposition and environmental triggers

Genetic factors	Host factors	Environmental factors
• Complement gene polymorphism	• Hypertension	• Exposure to sunlight
• Caucasian race	• Hyperopia	• Smoking
• Aging	• Blue eyes	• Obesity
• Female gender	• Nuclear cataract	• Nutrition

- Early in the disease, there are deposits in the Bruch's membrane due to a failure of the RPE to clear the debris
- This is followed by lipid deposits that are clinically visible as drusen
- Following genetic and environmental insults, there is a thickening of the Bruch's membrane due to deposition of calcium, increased levels of glycation end products and there is degeneration of collagen
- As a result, there is relative hypoxia of the outer retinal layers.
- This stimulates the formation of new vessels that lead to neovascular ARMD

C. Stages and Clinical Features

1. Dry ARMD: Early

- Early ARMD is characterized by the formation of drusen
- They are abnormal material deposited between the RPE and Bruch's membrane which occur due to a failure to be phagocytosed by the RPE
- They appear as yellow subretinal deposits and resemble hard exudates
- They may have sharp borders (hard drusen) or fuzzy borders (soft drusen)
- They may be small (<63 μm), intermediate (64–123 μm), and large (>124 μm)
- Progression is faster with soft and large drusen. Drusen may be a part of heredomacular degeneration called familial drusen and it is not age-related

2. **Dry ARMD: Advanced**
   - The advanced form is called geographic atrophy where there is severe visual morbidity
   - Fundus examination reveals a well-demarcated geographic area of RPE atrophy evidenced by thinned out retina and choroid with the visible underlying sclera
3. **Wet ARMD or neovascular ARMD**

Stage	Clinical features
Stage of pigment epithelial detachment • Due to impediment of hydraulic movement through the Bruch's membrane resulting in a collection of fluid below RPE	A sharply demarcated circular lesion, usually brown FFA reveals the pooling of dye which does not increase in size with time
Stage of choroidal neovascularization • Following a break in the Bruch's membrane, new vessels enter the sub RPE space (type 1) and subretinal space (type 2) to form a vascular membrane	Metamorphopsia, blurred vision, positive scotoma Sub RPE CNVM: Grayish elevated lesion with indistinct margins Subretinal CNVM appears like a subretinal halo with fluid in the subretinal space. It can be extrafoveal, juxtafoveal, or subfoveal Based on FFA it is described as classic, occult, or combined type
Stage of a hemorrhagic detachment of RPE • There is bleeding within the pigment epithelial detachment	Appears as a dark elevated mound at the macula
Stage of disciform scar • It occurs following a hemorrhagic episode	It appears as a yellowish subretinal scar

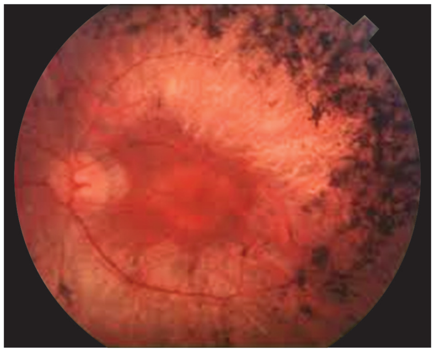
6. A 13-year-old boy is brought to an ophthalmologist by his parents. They feel he is finding it difficult to play outdoors during the evenings and prefers bright surroundings. Fundus examination shows the following image.

A. State the most probable diagnosis. (1 mark)

B. List three classical features that define this disease. (3 marks)

C. Describe the investigations that are helpful in follow-up of this condition. (3 marks)

D. Enumerate three more hereditary chorioretinal dystrophies. (3 marks)



A. Most Probable Diagnosis

Retinitis pigmentosa

B. Classical Features

1. Arteriolar attenuation
2. Bone-spicule pigmentation surrounding the veins
3. Waxy pallor of the optic disc

C. Investigations

1. **Dark adaptometry**
   - It is a sensitive test used for measuring the dark adaptation time. Also, it measures the visual threshold which is the minimum intensity of light that can elicit a rod response
   - In RP, the threshold is an increased rod threshold and prolonged dark adaptation time

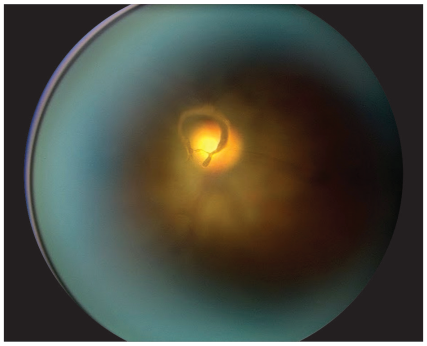
2. **Visual field**
   - Annular or ring-shaped scotoma is a characteristic feature of retinitis pigmentosa
   - As the disease progresses, the scotoma widens anteriorly and posteriorly and finally, the patient is left with a central tunnel or a tubular vision
3. **Electroretinogram**
   - Full-field ERG shows decreased scotopic and combined response. With further progression of the disease, there is a decrease in the photopic responses as well
   - Finally, the ERG responses are extinguished
4. **Optical coherence tomography**
   - It helps in confirming the presence of cystoid macular edema, a common association of retinitis pigmentosa.
   - It will also help to know the progress of CME following treatment
5. **Microperimetry**
   - It is a rapid, safe, and noninvasive technique in which the retinal sensitivity is measured while fundus is directly examined
   - It is especially helpful when the patient is unable to fixate and perform conventional perimetry
6. **Electrooculogram**
   - Absence of light peak
7. **Exome sequencing**
   - This helps in genetic counseling only.
   - It does not help in understanding the progress of the disease

D. Hereditary Chorioretinal Dystrophies

1. Leber's congenital amaurosis
2. Gyrate atrophy
3. Choroideremia

7. A 50-year-old lady presents with sudden onset of floaters in her right eye. On examination, the following image is seen floating anterior to the disc.

A. State the most probable diagnosis? (1 mark)
B. Name three factors that predispose to this condition. (3 marks)
C. Explain the management of this condition (3 marks)
D. Explain the pathophysiology of rhegmatogenous retinal detachment and its presenting features? (3 marks)



A. Most Probable Diagnosis

Posterior vitreous detachment (PVD).

B. Factors Predisposing to PVD

1. Age
2. Myopia
3. Trauma

C. Management of PVD

- Posterior vitreous detachment may be associated with the formation of peripheral retail tears

- Hence, it should be urgently followed up by a dilated fundus examination to look for the same especially in cases of myopes, family history of retinal detachment, pseudophakic, and Stickler syndrome

Evaluation

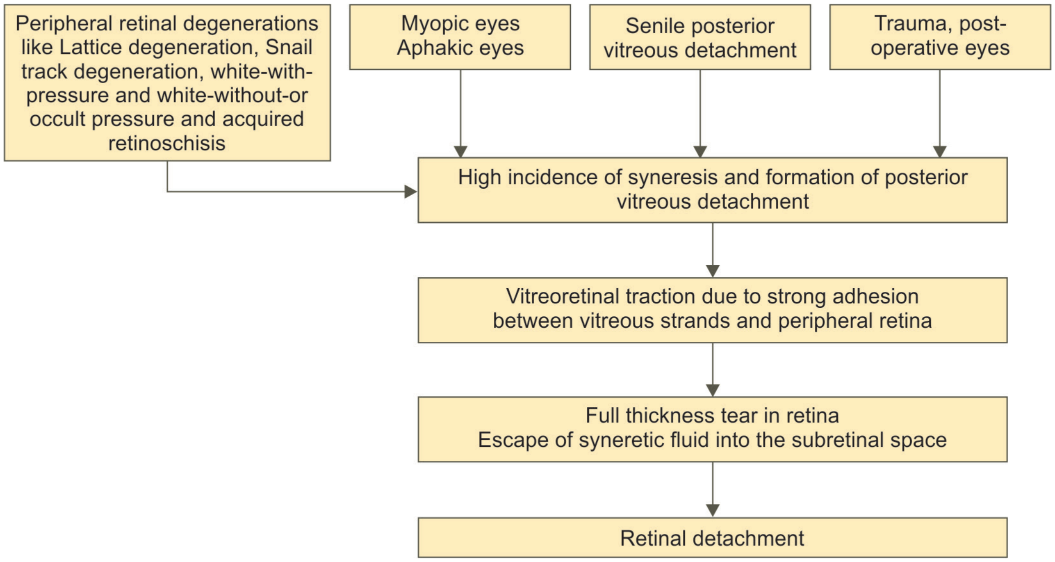
- The examination should be done to examine 360° of the retina till the ora serrata
- The fellow eye should also be examined even though asymptomatic
- The patient is educated thoroughly about the symptoms and signs of a retinal detachment like an increase in the size and number of floaters, associated photopsia, and decreased visual acuity/field.
- He should be educated about the urgency of the detachment

Specific Management

- There is no active management required for a PVD and the patients learn to ignore the cobweb-shaped floaters
- If PVD is associated with vitreous hemorrhage, the eye should be followed up by serial ultrasound closely to rule out underlying retinal detachment
- If associated with retinal breaks/tears, they should be treated with LASER photocoagulation

Tears needing urgent treatment	**Tears that can be conservatively managed**
• Symptomatic tears • Large breaks • Operculated tear • History of retinal detachment in the fellow eye, myopia, family history, Marfan syndrome, Stickler syndrome • U-shaped tears • Traumatic tears • Retinal dialysis • Subclinical retinal detachment	Round holes Asymptomatic U-tears

D. Pathophysiology of Rhegmatogenous Retinal Detachment and Clinical Features


Peripheral retinal degenerations like Lattice degeneration, Snail track degeneration, white-with-pressure and white-without-or occult pressure and acquired retinoschisis
Myopic eyes
Aphakic eyes
Senile posterior vitreous detachment
Trauma, post-operative eyes
High incidence of syneresis and formation of posterior vitreous detachment
Vitreoretinal traction due to strong adhesion between vitreous strands and peripheral retina
Full thickness tear in retina
Escape of syneretic fluid into the subretinal space
Retinal detachment

8. An 18-month-old child was brought with white reflex in the pupils of both eyes.

A. What is this condition called? (1 mark)
B. Name four differential diagnoses for the same. (2 marks)
C. Describe the genetics of retinoblastoma. (5 marks)
D. Name the four clinical stages of retinoblastoma. (2 marks)

A. Condition is

Leukocoria

B. Differential Diagnosis for Leukocoria

1. Congenital cataract
2. Retinoblastoma
3. Persistent hyperplastic primary vitreous
4. Coats' disease

C. Genetics of Retinoblastoma

- It is caused by mutation of the RB1 gene, which is a tumor suppressor gene located on the long arm of the 13th chromosome, locus 14. Both the copies must be mutated for the disease to manifest
- Kundson's two hit hypothesis explains the inheritance of retinoblastoma (Fig. 8.4.2)
  - One 'hit' or mutation is acquired genetically from one of the parents and the second 'hit' occurs as a somatic mutation in the postzygotic stage
  - Hence, at a molecular level, it behaves as a recessive disorder but clinically, it behaves as a dominant disorder

1. **Hereditary**
   - About 40% of all Rb cases are hereditary and bilateral tumors are *always hereditary*
   - They tend to be multifocal (trilateral retinoblastoma: Bilateral retinoblastoma with pineoblastoma)
   - If the parent has bilateral disease, there is a 45% chance of the offspring being affected, and 85% of this will be bilateral
     - There is a 45% chance that the sibling will also inherit the tumor

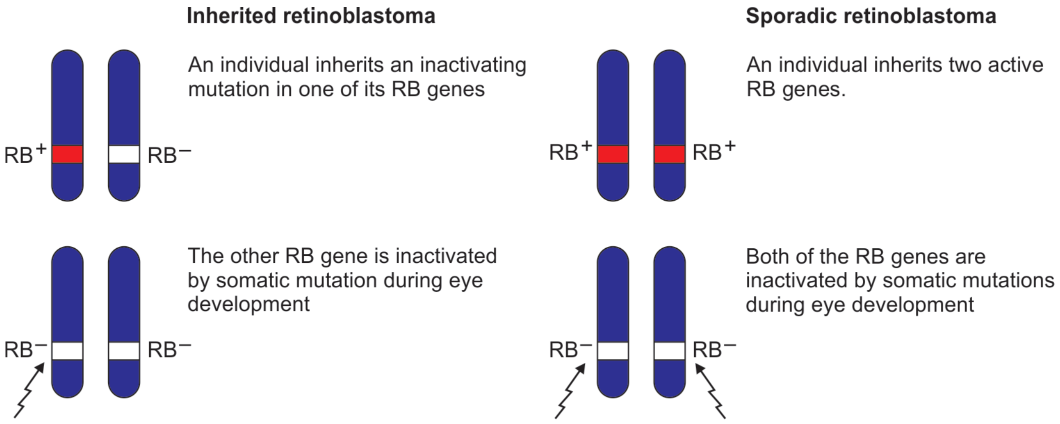

Inherited retinoblastoma
An individual inherits an inactivating mutation in one of its RB genes
RB+
RB−
The other RB gene is inactivated by somatic mutation during eye development
RB−
RB−
Sporadic retinoblastoma
An individual inherits two active RB genes.
RB+
RB+
Both of the RB genes are inactivated by somatic mutations during eye development
RB−
RB−

Fig. 8.4.2: Knudson's two-hit hypothesis for retinoblastoma

  - If the parent has unilateral disease, there is a 15% chance of acquiring the tumor with an 85% chance of it being bilateral
  - If the parent is a carrier, <1% chance of the offspring getting the tumor
2. **Sporadic**
  - Accounts for 60% of all cases of Rb
  - They are always unilateral and unifocal and the tumor is not transmissible

D. Clinical Stages of Retinoblastoma

1. Quiescent stage
2. Glaucomatous stage
3. Orbital retinoblastoma
4. Metastatic retinoblastoma

SHORT ESSAYS

1. Describe the grades of hypertensive retinopathy with their systemic implications. (5 marks)

Grading of Hypertensive Retinopathy (Keith and Wagner Classification)

- This classification indicates the chronicity of hypertension
- It is an independent indicator of the long-term risk of development of stroke and cerebral infarction, even if the blood pressure is normal

Grade	Description	Systemic Association
I	Mild generalized arteriolar attenuation, particularly of small branches, with a broadening of the arteriolar light reflex and vein concealment	Weak association with stroke, coronary artery disease, and cardiovascular mortality
II	Marked generalized narrowing and focal attenuation of arterioles associated with deflection of veins at arteriovenous crossings (Salus' sign) This happens as the vein tries to escape the arterial pressure by changing its direction	Strong association with stroke, congestive heart failure, renal dysfunction, and cardiovascular mortality
III	There is marked attenuation of arterioles + banking of veins distal to arteriovenous crossings (Bonnet sign), tapering of veins on either side of the crossings (Gunn sign) Flame-shaped hemorrhages cotton-wool spots and hard exudates are also present	Associated with mortality and renal failure
IV	This consists of all changes of grade III along with disc edema	

2. Describe the treatment of diabetic maculopathy. (5 marks)

Treatment of Diabetic Maculopathy

1. **Intravitreal injection of anti-VEGF agents**
  - Drugs like bevacizumab, ranibizumab, and aflibercept are becoming the first therapeutic agents of choice for diabetic macular edema. They are best suited for center-involving edema
  - However, repeat injections are needed. They are followed up with OCT
2. **Laser photocoagulation**
  - The use of laser photocoagulation was the standard of care for diabetic macular edema. FFA performed before laser helps to decide on the type of treatment

  - Diffuse edema is benefited by Grid laser photocoagulation (laser burns of 100 μm applied not closer than 500 μm from the center) whereas focal edema is benefited from focal laser application on the leaking microaneurysms. The spot size is smaller (50–100 μm at the leaking microaneurysms or to the center of the ring of hard exudates)

3. **Subthreshold (micropulse) diode laser**
   - Very short duration laser pulse with longer interval is more helpful as the collateral damage to the retina and choroid whilst stimulating the RPE
   - Subfoveal therapy is also possible
4. **Intravitreal Injection of steroid (triamcinolone)/fluocinolone implant**
   - It is best suited for pseudophakic eyes when the intravitreal injection is followed up with LASER
   - It is a good option in pregnancy when the use of anti-VEGF agents is contraindicated (as in pregnancy, recent thromboembolic events)
5. **Pars plana vitrectomy**
   - It is indicated when the edema is associated with tangential traction of the macula and a taut posterior hyaloid
   - To summarize

Macular edema involving the center, visual acuity better than 6/15	Intravitreal anti-VEGF agents or subthreshold LASER
Macular edema involving the center, visual acuity worse than 6/15 + significant foveal thickening	Intravitreal anti-VEGF agents followed by focal laser after 5–6 months. Intravitreal triamcinolone can be used if resistant to anti-VEGF agents
Macular edema not involving the center	Focal or grid or modified grid laser or Intravitreal anti-VEGF agents or triamcinolone
Macular edema with proliferative diabetic retinopathy	Anti-VEGF agents + PRP
Ischemic maculopathy	Poor prognosis, difficult to treat

3. Explain the role of optical coherence tomography (OCT) and fundus fluorescein angiography (FFA) in diabetic retinopathy. (5 marks)

Role of OCT

- It mainly helps in evaluating the macula for edema in diabetic retinopathy
- Macular edema can be classified as spongy edema, cystoid edema, and serous foveal detachment
  - Spongy edema is characterized by the presence of sponge-like retinal swelling
  - Cystoid edema is characterized by intraretinal cystoid spaces
  - Foveal detachment is characterized by the separation of the fovea from the RPE
- OCT helps to delineate the type of macular edema. Also, it demonstrates the thickness of the fovea as central subfield thickness in microns
- A thin fovea is a result of ischemia whereas thickness implies edema
- OCT also helps to follow-up patients with macular edema in response to various types of treatment like LASER and anti-VEGF agents
- Other macular pathologies like the presence of vitreomacular traction bands and epiretinal membranes can also be studied with the help of OCT

Role of FFA

It is a prerequisite to LASER treatment in PRP as well as focal

- In PDR, it highlights new vessels which show hyperfluorescence. It helps to differentiate IRMA from new vessels
- It also helps to distinguish between dot/ blot hemorrhages and microaneurysms
- FA helps in identifying areas of capillary nonperfusion
- In macular edema, it helps to distinguish the two main types of macular edema, focal and diffuse
- It helps to diagnose ischemic maculopathy which is a diagnosis by FA alone. Ischemic maculopathy is defined by an enlarged foveal avascular zone and carries a poor prognosis

4. Describe the clinical features of sickle cell retinopathy. (5 marks)

- Sickle cell anemia is an abnormal hemoglobinopathy characterized by sickling on an RBC under hypoxic conditions
- They are more fragile and do not possess the 'elasticity' of normal RBCs. As a result, they are capable of occluding small vessels

Clinical Features

- In the anterior segment, isolated dark comma-shaped anomalous conjunctival vessels may be seen
- Iris may show atrophic patches due to segmental ischemia
- Fundus changes that are associated with sickling are divided into nonproliferative and proliferative retinopathy

Nonproliferative changes	Proliferative retinopathy
• Venous tortuosity (due to stagnation of blood flow) • Silver wiring of peripheral vessels (previously occluded) • Black sunbursts: Areas of focal RE hyperplasia • Salmon patches: Pink intraretinal hemorrhages • Macular depression sign (due to thinning)	• Stage of arteriolar obstruction • Stage of arteriovenous anastomosis • Stage of neovascularization: It is called a 'sea fan' appearance. New vessels arise as a tuft of fine vessels, most of which regress spontaneously due to autoinfarction. They appear as grayish bands • Stage of vitreous hemorrhage • Stage of tractional retinal detachment

5. Describe the zones and stages of retinopathy of prematurity. (5 marks)

Zones of ROP (Fig. 8.4.3)

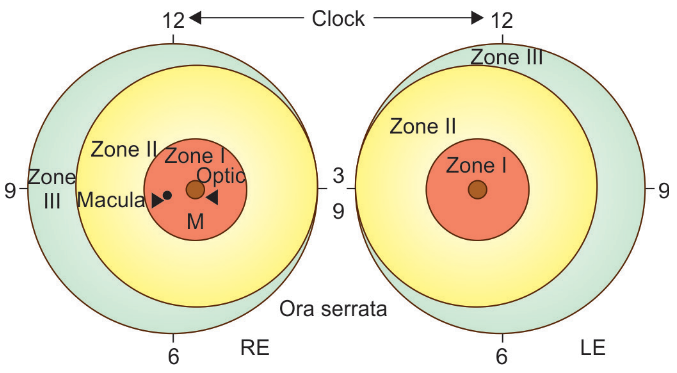


Fig. 8.4.3: Zones of ROP

1. **Zone 1**
    - Twice the distance between the macula and the optic nerve is taken as the radius and a circle is drawn with the disc as the center
2. **Zone 2**
    - The radius is taken as the distance from the macula and nasal ora serrata and a circle is drawn concentric to the previous circle
3. **Zone 3**
    - Remaining temporal retina
    - The extent of ROP is described in the number of clock hours of involvement

Stages of ROP (Fig. 8.4.4)

1. **Stage 1**
    - Formation of a demarcation line between vascularized and avascular retina
2. **Stage 2**
    - The line thickens to form a ridge
    - The vessels are seen entering the ridge but not above it
3. **Stage 3**
    - New vessels are projecting into the vitreous from the ridge
4. **Stage 4**
    - Partial retinal detachment: a—extrafoveal, b—foveal
5. **Stage 5**
    - Total retinal detachment

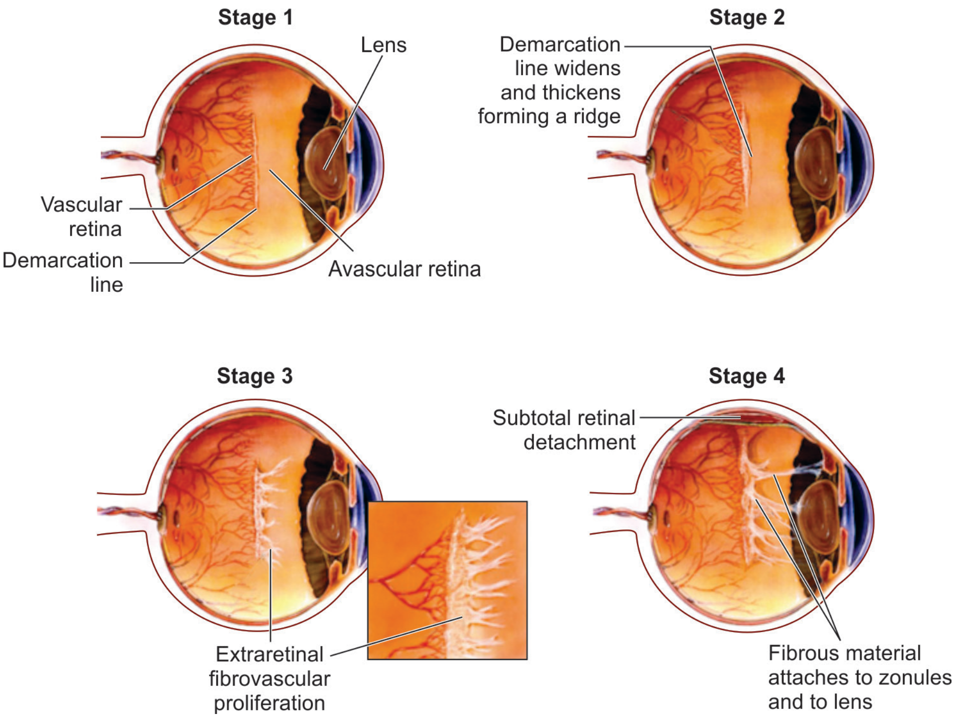

Stage 1
Lens
Vascular retina
Demarcation line
Avascular retina
Stage 2
Demarcation line widens and thickens forming a ridge
Stage 3
Extraretinal fibrovascular proliferation
Stage 4
Subtotal retinal detachment
Fibrous material attaches to zonules and to lens

Fig. 8.4.4: Stages of ROP

6. Describe the follow-up and treatment of ROP. (5 marks)

Treatment depends on the stage of ROP and the zone of the retina that is affected

Stage IVA	Pars plana vitrectomy
Threshold disease Presence of stage 3 with plus disease in zones I or II involving 5 contiguous clock hours or 8 noncontiguous clock hours	Laser ablation of the avascular retina with argon laser
Prethreshold disease type I Any ROP in zone I accompanied by plus disease or Stage 3 to any extent in zone I or Stage 2 or 3 in zone II with plus disease	Laser ablation of the avascular retinal with argon laser
Rush disease ROP in zone I with aggressive plus disease	Laser ablation of the avascular retinal with argon laser
Prethreshold disease type II Stage 1 or 2 in zone 1 without plus disease Stage 3 ROP in zone II without plus disease	Observation Weekly follow-up
Stage 1 or 2 ROP in zone I Stage 3 ROP in zone II	Weekly follow-up
Immature retina in zone I Stage 2 in zone II Regressing ROP in zone I	1–2 weeks follow up
Stage 1 ROP in zone II Regressing ROP in zone II	2 weeks follow-up
Stage 1 or 2 in zone III Regressing ROP in zone III	2–3 weeks follow-up

The infants are followed up till they complete 40 weeks of gestation or till vasculogenesis is complete, whichever is later

- The role of anti-VEGF agents is being explored.
- Their dosage, efficacy, and safety guidelines have yet to be established

7. Describe the clinical features and management of Coats' disease. (2+3 marks)

Coats' Disease

A disorder of childhood that is characterized by idiopathic retinal telangiectasias.

Clinical Features

- Presentation is usually in the first or second decade with unilateral painless visual loss. Some parents may report leukocoria
- Some children may present with strabismus
- Fundus examination shows telangiectasias and fusiform focal aneurysmal dilatation of arterioles more in the inferior and temporal quadrants in the preequatorial regions
- This is followed by intraretinal and subretinal exudation. It may progress to exudative retinal detachment
- Complications include neovascularization, neovascular glaucoma, cataract, and phthisis bulbi

Management

- FFA and OCT can be used to quantify the amount of macular edema
- Depends on the stage and presentation of the disease. It can be observed and followed up when the aneurysms are in the periphery and cause minimal exudation
- If exudation is excessive, the leakage points can be ablated with a laser
- Intravitreal anti-VEGF agents can be given to decrease the exudation
- Intravitreal triamcinolone can be used to decrease exudation in exudative retinal detachment
- In presence of tractional bands and retinal detachment, vitreoretinal surgery is performed
- In cases with neovascular glaucoma with no visual potential, enucleation is performed

8. Describe the clinical features and management of Eales' disease. (2+3 marks)

Eales' Disease/Periphlebitis Retinae

An idiopathic inflammatory disorder of the retinal vessels characterized by occlusive periphlebitis.

Clinical Features

- It is seen in young males, and more in the Indian subcontinent
- It is a common cause of spontaneous vitreous hemorrhage and as a result, the disease is silent till the patient develops floaters and diminution of vision due to hemorrhage
- On examination, there may be mild anterior uveitis
- Fundus examination demonstrates perivenous exudation in the peripheral retina along with superficial hemorrhages and sometimes cotton wool spots. This is the inflammatory phase of the disease
- Later, microaneurysms, vascular tortuosity, shunts, nonperfusion areas, and neovascularization develop. Neovascularization develops at the junction of perfused and non-perfused areas and they tend to bleed spontaneously
- Vitreous hemorrhage is usually self-limiting

Management

- Corticosteroids are the mainstay of treatment: In the form of topical, periocular, intravitreal, and systemic routes
- Antitubercular therapy may be considered if supported by positive tests for tuberculosis like quantiferon gamma
- Photocoagulation of the avascular areas is required to prevent the formation of new vessels
- Persistent vitreous hemorrhage requires a vitrectomy

9. Describe the clinical features of macular disorders. (5 marks)

Macular Disorders

- The macula is very important for central vision and its functioning depends on the arrangement of cones
- When there is a macular pathology, this arrangement is disturbed

Clinical Features

Symptoms of macular disorder

- Metamorphopsia: It is described as distorted vision due to a change in the arrangement of the photoreceptors. It is very specific for macular diseases
- Micropsia: The appearance of an object smaller than its actual size happens due to an increase in the distance between two cones
- Macropsia: The appearance of an object larger than its actual size happens due to a decrease in the distance between two cones (crowding)

Signs of macular disorder

- Visual acuity is reduced. It may range from 6/6 to 6/60. In macular pathologies, peripheral vision is spared. Near vision is worse affected
- Color vision defects: blue color is the worst affected
- Fundus examination shows abnormalities like elevation, loss of foveal reflex, wrinkling, deposits like drusen or hard exudates, and thickening. It is best appreciated using slit-lamp biomicroscopy
- Photostress recovery time
  - It is a clinical subjective test performed by shining a bright source of light on the involved eye after noting the best-corrected visual acuity
  - The light is shone for 10 seconds
  - The patient's visual acuity is noted after the procedure. Usually, the patient should be able to read the same line before 30 seconds
  - Anything more than 45 seconds is a delayed photostress time
- Central positive scotoma is detected on an Amsler grid and visual field testing

10. Describe the etiology, clinical features, and management of central serous chorioretinopathy (CSCR). (1+2+2 marks)

Central Serous Chorioretinopathy (CSCR)

Etiology

- It is an idiopathic disorder characterized by hyperpermeability of the RPE and leakage from the choriocapillaris
- Various risk factors have been identified like male gender, hypermetropia, type A personality, use of systemic

Clinical Features

Symptoms

- Acute onset of blurred vision
- Metamorphopsia
- Micropsia
- Macropsia
- Positive scotoma

Signs

- Visual acuity is decreased or maybe normal
- Usually, it is correctable by the use of convex lenses as the retina is elevated
- The anterior chamber is normal

- Amsler chart shows a central/paracentral scotoma
- Fundus shows an elevated, circular lesion in the macular area
- It is usually self-limiting, although in some cases there might be recurrences

Management

- OCT merely helps in confirmation as the diagnosis is mainly clinical. FFA helps to locate the sites of the leak
- Most of the time, it is self-limiting
- Treatment is needed if
  - Serous detachment persists >3 months
  - Recurrence of the disease in the eye has visual morbidity due to the same
  - A permanent visual deficit is present in the fellow eye due to CSCR
  - Occupational requirement
- Choices of treatment available are:
  - Laser photocoagulation at the site of the leak in FFA
  - Photodynamic therapy when the source is very close to the fovea
  - Intravitreal anti-VEGF agents

11. Describe the investigations useful in central serous chorioretinopathy.

(5 marks)

1. **Fundus fluorescein angiography**
   - Demonstrates a leak in the arterial phase which increases in intensity and size with time
   - The leak is due to dye pooling into the subretinal space
   - It is called the expansile dot pattern and more commonly seen
   - Another pattern is the smokestack pattern, wherein the dye appears as a dot and then slowly moves up vertically like a rising plume of smoke
   - It is due to the convection currents and due to protein concentration gradient between subretinal fluid and the fluorescein dye
   - This unique pattern is however seen only in 10% of the cases
2. **Optical coherence tomography**
   - It helps to find out subtle leaks, if not visible on fundus examination, and understand the location of the leak in terms of layers of the retina
   - A subretinal leak is seen as a less convex collection of fluid, beneath the RPE
   - Whereas a pigment epithelial detachment is seen as more convex and is deeper
3. **Fundus autofluorescence**
   - Focal decrease in the autofluorescence at the site of leakage, both active and old
4. **ICG angiography**
   - It demonstrates dilated and compromised choroidal vessels at the posterior pole and mid-stage areas of hyperfluorescence
   - It helps to detect subclinical foci also

12. Describe the causes and clinical features of cystoid macular edema. Outline the management based on etiology. (5 marks)

Cystoid Macular Edema

Etiology

Vascular	Inflammation	Drugs	Traction
• Diabetic macular edema • Retinal vein occlusion • Retinal telangiectasias • Radiation retinopathy	• Anterior uveitis • Intermediate uveitis • Posterior uveitis • Following cataract surgery (especially ICCE and when there is a breach in the posterior capsule) • Following Nd: YAG capsulotomy, PRP	• Epinephrine • Prostaglandins • Systemic nicotinic acid	• Vitreomacular traction

Clinical Features

- They present with metamorphopsia, central scotoma, and decreased vision to about 6/60
- Fundus examination reveals honeycomb-like elevation of the macula
- Amsler grid demonstrates metamorphopsia
- FFA shows flower petal appearance which is pathognomonic for CME
- OCT shows cystoid spaces in the inner nuclear and outer plexiform layers

Management

Etiology	Management
• Vascular cause	• LASER photocoagulation, anti-VEGF injections
• Inflammatory	• Topical NSAIDs, Topical steroids, periocular steroids–intravitreal steroids/antiVEGF
• Following cataract surgery	• Topical or periocular steroids, pars plana vitrectomy can be done
• Following LASER procedures	• Unsatisfactory
• Associated with RP and other dystrophies	• Systemic carbonic anhydrase inhibitors
• Vitreomacular traction	• Vitrectomy

13. Describe the clinical features and management of neovascular age-related macular degeneration (ARMD). (5 marks)

Neovascular ARMD

Stage	Clinical features
• Stage of pigment epithelial detachment • Due to impediment of hydraulic movement through the Bruch's membrane resulting in a collection of fluid below RPE	• Sharply demarcated circular lesion, usually brown • FFA reveals the pooling of dye which does not increase in size with time • No treatment is required • It may progress to an RPE tear

Contd.

Stage	Clinical features
• Stage of choroidal neovascularization • Following a break in the Bruch's membrane, new vessels enter the sub RPE space (type 1) and subretinal space (type 2) to form a vascular membrane	• Metamorphopsia, blurred vision, positive scotoma • Sub RPE CNVM: Grayish elevated lesion with indistinct margins • Subretinal CNVM: Appears like a subretinal halo with fluid in the subretinal space • It can be extrafoveal, juxtafoveal, or subfoveal • Based on FFA it is described as classic, occult, or combined type
• Stage of a hemorrhagic detachment of RPE • There is bleeding within the pigment epithelial detachment	• Appears as a dark elevated mound at the macula
• Stage of disciform scar • It occurs following a hemorrhagic episode	• It appears as a yellowish subretinal scar

Neovascular or exudative ARMD progresses through various stages.

Management

Pigment epithelial detachment	• Observation • Intravitreal anti-VEGF in cases of associated CNVM
Choroidal neovascular membrane	• FFA and OCT are used to classify choroidal neovascular membrane • Based on whether the neovascular complex is present in the sub-RPE space or subretinal space, it is classified as type I and type II • Based on FFA, CNVM is classified as classic and occult. Classic CNVM (type II) is defined as a well-defined lesion with a lacy pattern in the early stages, with pooling in the subretinal space and late staining of the fibrous tissue • Occult CNVM has irregular borders and filling with late filling and staining • All subtypes of CNVM respond well to intravitreal anti-VEGF agents • Photodynamic therapy with Verteporfin is useful in subfoveal predominantly classic types of CNVM • LASER photocoagulation is useful in extrafoveal CNVM
Hemorrhagic detachment of RPE	• Poor prognosis
Disciform scar	• Poor prognosis

14. Explain the technique of an intravitreal injection. Name one adverse reaction to intravitreal triamcinolone. (4+1 marks)

Technique of an Intravitreal Injection

- The intravitreal injection has the advantage of delivering the right amount of drug to the retina and choroid. Many drugs like antibiotics, anti-VEGF agents, steroids, antivirals, and antifungals are being delivered via this route
- The procedure can be done on an outpatient basis but in a sterile atmosphere. Surface ocular infections should be ruled out by meticulous slit-lamp examination, sac syringing. IOP should be checked
- The conjunctival sac is cleaned with povidone-iodine and a topical anesthetic is instilled. Wire speculum is placed

- A preloaded syringe containing the necessary drug with the appropriate dose is kept ready. The inferior quadrant is chosen due to the ease of injection
- The patient is instructed to maintain a straight gaze. A caliper is used to mark 3.5 mm behind the limbus in pseudophakic eyes and 4mm behind the limbus in phakic eyes
- The globe is stabilized at the opposite limbus with the help of forceps. The needle of the syringe is entered perpendicular to the globe through the conjunctiva with the beveled edge towards the surgeon
- The direction of the needle should be towards the center of the globe. The drug is injected
- The needle is carefully withdrawn and discarded

Adverse Reaction to Intravitreal Triamcinolone

Rises in intraocular pressure.

15. A 22-year-old-man complains of seeing a ring in the center of his visual field after he bet to watch the solar eclipse. Explain the pathophysiology, fundus features, and prognosis of this condition. (5 marks)

The most probable condition that follows the given scenario is solar retinopathy.

Solar Retinopathy

Pathophysiology

- Viewing the sun for >90 seconds through a 3 mm pupil rises the retinal temperature by 4°C. if the pupil is dilated, it rises by 22°C
- This is well above photocoagulation temperature
- Due to this, there is a fragmentation of the photoreceptors

Fundus Features

- Patients typically present around 4 hours after exposure with distorted vision and central scotoma
- Visual acuity at presentation may vary from 6/60-6/12 and may improve to 6/12-6/6 after 6 months
- Fundus shows a well yellow spot at the fovea with a grey margin in the perifoveal region
- After a few hours, the yellow spot is replaced by a defect in the fovea with irregular borders

Prognosis

- Visual acuity returns to normal in most cases
- Paracentral scotoma and metamorphopsia may persist permanently in some patients despite a good visual acuity

16. Explain the fundus findings of retinitis pigmentosa. Name two ocular associations. (3+2 marks)

Fundus Findings of Retinitis Pigmentosa include

- The first sign to develop is arteriolar attenuation
- It is followed by bony spicule-like pigmentary clumps in the equatorial retina, especially perivenous
- Later, these spread towards the periphery and posterior pole

- Other features are tessellation of the fundus due to diffuse RPE atrophy
- Macula shows thinning or epiretinal membrane or cystoid macular edema
- The disc becomes waxy yellow due to consecutive optic atrophy

Ocular Associations

1. Posterior subcapsular cataract
2. Primary open-angle glaucoma

17. Describe the syndromic associations of retinitis pigmentosa. (5 marks)

Syndromic Associations of Retinitis Pigmentosa (RP)

1. **Lawrence moon biedl/bardet biedl syndrome**
    - RP + Obesity + Hypogonadism + Polydactyly + Mental deficiency
2. **Cockayne's syndrome**
    - RP + Progressive infantile deafness + Dwarfism + Mental retardation + Nystagmus + Ataxia
3. **Refsum's syndrome**
    - RP + Peripheral neuropathy + Cerebellar ataxia (due to phytanic acid deficiency – the only reversible form of RP)
4. **Usher's syndrome**
    - RP + Labyrinthine deafness
5. **Hallgren's syndrome**
    - RP + Vestibulo-cerebellar ataxia + Congenital deafness + Mental deficiency
6. **Kearns–Sayre syndrome**
    - Mitochondrial inheritance
    - RP + Progressive complete ophthalmoplegia
7. **Bassen Kronzweig syndrome**
    - RP + Abetalipoproteinemia
    - It is treated with vitamin E

18. Define retinal detachment. Describe its types and etiologies. (5 marks)

Retinal Detachment

Definition

The separation between the neurosensory retina and the pigment epithelium.

Types and Etiologies

Type	Causes
• Rhegmatogenous • The retinal detachment is due to the accumulation of syneretic fluid between the RPE and the neurosensory retina through a break or tears in the retina.	• Retinal holes, tears due to degeneration • Inflammation • Trauma • Associated myopia • Posterior vitreous detachment

Contd.

Type	Causes
• Exudative • It is caused either due to tumors or accumulation of fluid between the RPE and neurosensory retina	• Vogt–Koyanagi–Harada syndrome • Toxemia of pregnancy • Renal hypertension • Posterior scleritis • central serous chorioretinopathy • Intraocular tumors like retinoblastoma and melanomas • Hemangiomas
• Tractional • It is caused by tractional bands (usually fibrovascular bands)	• Formation of traction bands can occur following trauma • Vasculoproliferative disorders like diabetic retinopathy • CRVO < Eales' disease

19. Describe the utility of Linckoff's rules in rhegmatogenous retinal detachment. (5 marks)

Linkcoff's Rules

Are used to localize the break in the rhegmatogenous retinal detachment based on the configuration of the retinal detachment (Fig. 8.4.5)

1. Shallow inferior retinal detachment with SRF higher on one side: The break is inferior and is present on the same side as the higher side of SRF
2. Inferior retinal detachment with equal fluid levels: tear is at 6 o'clock positions
3. Inferior bullous retinal detachment: Tear lies above the horizontal meridian
4. Inferior RD with SRF high, revolving around the optic disc: Break is in either side of the upper quadrant
5. Sub-total RD with a superior wedge of the attached retina: The break is near the periphery of the higher border of SRF
6. Superior RD with SRF crossing the vertical midline: The break is near 12 o'clock, the lower edge of the RD corresponds to the side of the break

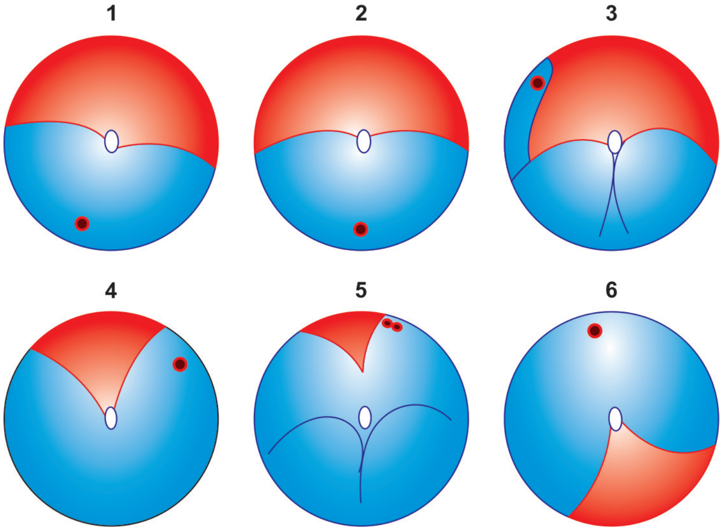


Fig. 8.4.5: Linckoff rules

20. Explain the method of retinal drawing with due importance to the color-coding. (5 marks)

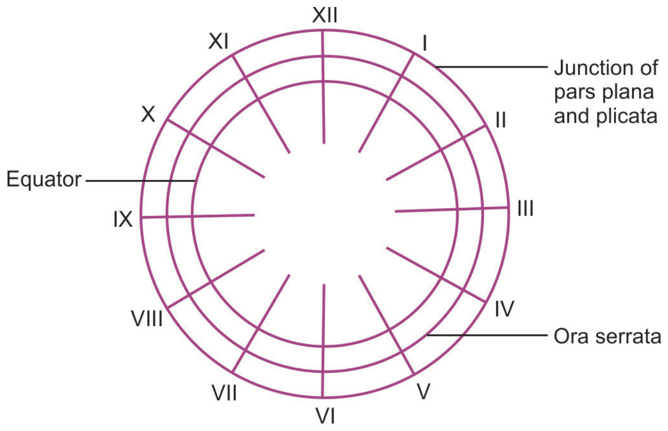


Fig. 8.4.6: Retinal drawing

- The method of retinal drawing is important to maintain uniformity in the documentation of retinal lesions
- This charting is known as Amsler–Dubois charting (Fig. 8.4.6)
- Three concentric circles are drawn. The outermost circle represents the junction of pars plana and ora serrata, the middle circle represents the ora serrata, and the innermost circle represents the equator
- 12 o’clock hours are marked intersecting all three lines. The center of the chat represents the macula and the side at which the optic disc is drawn represents the side of the eye.

Color-coding Used in Retinal Drawing

• Red color	• Hemorrhages • Attached retina • Arterioles • Vascular abnormalities • Tumors • Neovascularization
• Blue color	• Detached retina • Retina venules • Outlines of retinal breaks • Outline of lattice degeneration • Areas of white without pressure
• Green color	• Vitreous opacities • IOL • Intraocular foreign body • Hyaloid ring • Asteroid hyalosis • Lattice degeneration
• Brown color	• Uvea • Pigment epithelial detachment • Choroidal detachment • Choroidal melanomas
• Yellow color	• Hard exudates • Cotton wool spots • Exudation • Retinal edema • Drusen
• Black	• Laser marks • Sclerosed vessels • Pigments

21. Describe the fundus appearance of various types of retinal detachment. (5 marks)

Fundus Appearance of Various Types of Retinal Detachment

Rhegmatogenous retinal detachment	• The retinal detachment appears pale as it is elevated from the underlying choroid • It has a convex appearance and corrugated appearance due to edema. Retinal blood vessels appear darker. The highest point of retinal detachment is determined by the site of the break (usually on the higher side of SRF) • It is always associated with subretinal fluid, which may extend from the site of the break to the ora serrata. It does not change position significantly with a change in the posture of the patient • B-scan shows good mobility of the retina and vitreous
Exudative retinal detachment	• The configuration of the retinal detachment is convex and smooth (cf corrugated in rhegmatogenous RD) • The detached retina is very mobile and demonstrates shifting fluid
Tractional retinal detachment	• The retinal detachment has a concave configuration and there are no breaks • The highest point on the detached retina corresponds to the site of vitreoretinal traction • The SRF is shallower and does not extend till the ora serrata

22. Describe the principles of management of a rhegmatogenous retinal detachment. (5 marks)

Management of Rhegmatogenous Retinal Detachment

1. Finding the causative break/breaks by a thorough dilated fundus examination. The location of the break can be guessed by using Linckoff's rules based on the configuration of the RD
2. Sealing the break by creating aseptic chorioretinitis with cryotherapy or photocoagulation
3. Subretinal fluid (SRF) must be drained in long-standing RDs (as serous fluid becomes turbid) and deep SRF. It can be done by making a full-thickness sclerotomy after applying high pressure on the globe
4. To cause sclera-chorioretinal apposition
   - Scleral buckling
     - Silicone bands of varying arc lengths are placed under the conjunctiva over the sclera to create an inward indentation of the sclera to provide external tamponade
     - Radial explants are useful in isolated tears and circumferential bands in breaks in quadrants and large tears
   - Pneumoretinopexy
     - It is a simple outpatient procedure that is reserved for fresh superior RD with one or two small holes extending over less than two clock hours in the upper two-thirds of the peripheral retina
     - Here, the breaks are sealed first by cryotherapy followed by injection of a small amount of expansile gas like sulphur hexafluoride or perfluoropropane into the vitreous and positioning the patient so that the gas bubble remains in contact with the tear for 5–7 days
5. Pars plana vitrectomy
   - It is done for giant tears and the presence of PVR. It is becoming the treatment of choice in all types of rhegmatogenous retinal detachment

- It is done by pars plana vitrectomy followed by internal drainage of SRF through existing retinal breaks using a fine needle or through a posterior retinotomy is done
- Tamponade is achieved by injecting silicone oil or perfluorocarbons liquid
- Endolaser is then applied around the area of retinal tears to achieve adhesion

23. Describe the histopathology of retinoblastoma. (5 marks)

Histopathology of Retinoblastoma

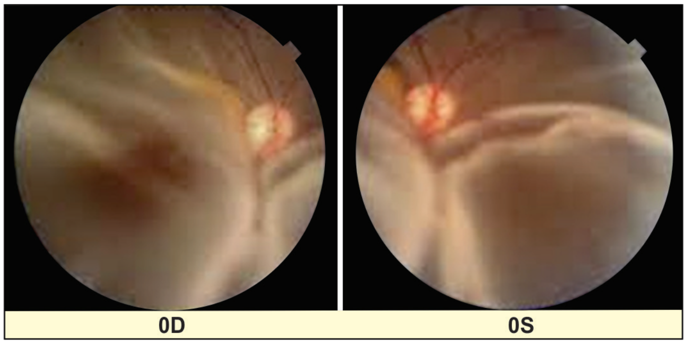
- Small round cells with large nuclei, resembling the cells of the nuclear layer of the retina
- These cells may present as a highly undifferentiated or well-differentiated tumor
- Microscopic features of a well-differentiated tumor are:
    1. *Flexner–Wintersteiner rosettes (highly specific of retinoblastoma):* A single layer of columnar cells around a clear lumen
    2. *Homer–Wright rosettes:* Cells arranged around a central fibrillary core (also seen in medulloepithelioma)
    3. *Pseudorosettes:* Tumor cells clustered around necrotic tissue
    4. *Fleurettes formation:* Very specific for Rb. Flower bouquet type of appearance of tumor cells
    5. *Other features* are calcification and necrosis

24. Describe the staging of retinoblastoma with management. (5 marks)

Staging of Retinoblastoma and Treatment Protocol

Staging	Treatment protocol
Group A: Intraretinal, small tumors (<3 mm in width, <2mm in thickness), away from macula and disc	Cryotherapy Transpupillary thermotherapy, or photocoagulation
Group B: Intraretinal tumors, of any size greater than group A but no vitreous seeding or subretinal seeding/fluid	Chemoreduction followed by focal therapy of brachyradiation
Group C: Discrete tumors, subretinal seeding <5mm from the tumor, minimal vitreous seeding	Chemoreduction followed by focal therapy of brachyradiation
Group D: Significant seeding, massive tumor, subretinal fluid causing retinal detachment	High dose chemotherapy followed by focal therapy followed by periocular carboplatin
Group E: Tumor touching lens, neovascular glaucoma, aseptic orbital cellulitis, phthisis bulbi, or diffuse infiltrating retinoblastoma	Enucleation followed by histopathological assessment for risk factors (undifferentiated or differentiated) Followed by 6 cycles of chemotherapy and external beam radiation and 12 cycles of chemotherapy Metastatic workup
Orbital retinoblastoma	High dose chemoreduction followed by enucleation, EBRT, and 12 cycles of chemotherapy
Lymph nodes involvement	Neck dissection → 6 cycles of chemotherapy → EBRT → 12 cycles of chemotherapy
Metastasis	Palliative – combination of chemotherapy + EBRT + exenteration

25. A 24-year-old primigravida with eclampsia complains of bilateral blurred vision of sudden onset. On examination, the following fundus finding is seen.



A. State the type of retinal detachment that is usually seen in association with PIH? (1 mark)

B. Describe the grades of hypertensive retinopathy associated with this condition. (2 marks)

C. Describe the management of this condition. (2 marks)

A. Type of Retinal Detachment

Exudative retinal detachment

B. Grades of Hypertensive Retinopathy Associated with PIH

Same as Keith and Wagener's classification

Grade I	Mild generalized arteriolar attenuation, particularly of small branches, with broadening of the arteriolar light reflex and vein concealment
Grade II	Marked generalized narrowing and focal attenuation of arterioles associated with deflection of veins at arteriovenous crossings (Salus' sign) This happens as the vein tries to escape the arterial pressure by changing its direction
Grade III	There is marked attenuation of arterioles + banking of veins distal to arteriovenous crossings (Bonnet sign), tapering of veins on either side of the crossings (Gunn sign) Flame-shaped hemorrhages cotton-wool spots and hard exudates are also present
Grade IV	This consists of all changes of grade III along with disc edema

C. Management

- The mainstay of management is control of blood pressure with the help of antihypertensives
- If the retinal detachment does not subside on its own, there may be associated retinal atrophy

26. Describe the effects of hypertension on the eye. (5 marks)

The manifestations of hypertension are retinopathy, choroidopathy, and optic neuropathy.

1. **Hypertensive retinopathy**
    - It refers to the retinal vascular response to systemic hypertension. As a result of an increase in systemic hypertension, there is focal and diffuse vasoconstriction
    - This is an acute response and is reversed when BP is controlled
    - When there is sustained hypertension, there is hypertrophy of the medial layer and hyalinization
    - Therefore, arteriosclerosis reflects chronic hypertension
    - When there is accelerated hypertension, there is an increase in vascular permeability due to the induced hypoxia. As a result, there is edema of the optic nerve head and exudation at the macula. Also, small vessels rupture under pressure and lead to hemorrhages
2. **Hypertensive choroidopathy**
    - It is manifested by Elschnig spots—focal areas of RPE necrosis due to occlusion of choriocapillaris
    - Siegrist streaks are linear flecks along choroidal vessels due to fibrinoid necrosis
    - Exudative retinal detachment also may occur
3. **Hypertensive optic neuropathy**
    - It is characterized by disc edema secondary to raised intracranial pressure because of hypertension

SHORT ANSWERS

1. Give the Keith and Wagener's grading for hypertensive retinopathy. (3 marks)

Grade I	Mild generalized arteriolar attenuation, particularly of small branches, with broadening of the arteriolar light reflex and vein concealment
Grade II	Marked generalized narrowing and focal attenuation of arterioles associated with deflection of veins at arteriovenous crossings (Salus' sign) This happens as the vein tries to escape the arterial pressure by changing its direction
Grade III	There is marked attenuation of arterioles + banking of veins distal to arteriovenous crossings (Bonnet sign), Tapering of veins on either side of the crossings (Gunn sign) Flame-shaped hemorrhages cotton-wool spots and hard exudates are also present
Grade IV	This consists of all changes of grade III along with disc edema

2. Describe the fundus features of malignant hypertension. (3 marks)

- Malignant hypertension is described as systolic blood pressure above 200 mm Hg and diastolic pressure more than 130 mm Hg
- Features are bilateral
- Fundus features
    - Disc edema
    - Shows severe arteriolar narrowing
    - Cotton wool spots
    - Intraretinal transudates
    - Macular edema in the form exudates around the macula simulating a star
    - Peripapillary retinal hemorrhages
- Edema is disproportionately greater than other retinal findings. It happens due to severe hypoxia.

3. Explain (1 mark each)

A. Bonnet's sign
B. Salu's sign
C. Gunn sign

A. Bonnet's Sign

- It is the banking of the veins on both sides of the artery crossing it
- It is seen in grade III hypertensive retinopathy

B. Salu's Sign

- Deflection of veins at arteriovenous crossings
- It is seen in grade II hypertensive retinopathy

C. Gunn Sign

- Tapering of veins on either side of the arteriovenous crossings
- It is seen in grade III hypertensive retinopathy

4. Describe the grading of arteriolosclerosis in the retina. (3 marks)

- It is seen in the elderly, where there is involutional sclerosis of the arteries
- The arteries are so thin that they are transparent. Hence the column of blood is seen through it
- In sclerosis, this transparency slowly gets replaced by opacification

Grade 1	Subtle broadening of arteriolar light reflex
Grade 2	Marked broadening of light reflex
Grade 3	Copper wiring of arteries
Grade 4	Silver wiring of arteries

5. Enumerate the risk factors responsible for the development and progression of diabetic retinopathy. (3 marks)

1. Duration of diabetes—*Most important*
2. Patients with type 1 diabetes are at a greater risk than type 2. They develop retinopathy in about 15 years
3. Poor glycemic control
4. Pregnancy
5. Coexisting hypertension, hyperlipidemia, renal pathology, anemia, and other vascular disorders
6. Other risk factors: Hyperlipidemia, smoking, cataract, obesity

6. Define clinically significant macular edema. (3 marks)

Clinically significant macular edema is defined as

- Presence of macular thickening within 500 μm from the foveola
- Presence of hard exudates within 500 μm from the foveola
- Presence of hard exudates of 1500 μm size or larger, a part of which is within 500 μm of foveola

7. Define ischemic maculopathy. Explain its management. (3 marks)

Ischemic Maculopathy

Definition

- Based on fluorescein angiography, as enlargement of foveal avascular zone 500 µm and changes in the contour of macular capillaries (irregular and widely spaced)
- Based on optical coherence tomography, ischemic maculopathy is defined as a decrease in the thickness of the nerve fiber layer in the macula

Management

- There is no defined treatment for ischemic maculopathy
- Control of blood sugars and blood pressure
- Grid laser, intravitreal triamcinolone, and intravitreal anti-VEGF agents are treatment options with no proven benefits

8. Enumerate three conditions that lead to retinal neovascularization. (3 marks)

1. Diabetic retinopathy
2. Retinal venous occlusions
3. Coats' disease

9. Define

A. Threshold ROP
B. Rush disease
C. Prethreshold type I ROP

A. Threshold Disease

Stage 3+ in zones I or II involving 5 contiguous clock hours or 8 noncontiguous clock hours

B. Rush Disease

- Also known as aggressive posterior ROP
- The disease is located at the posterior pole, characterized by rapid progression to stage 5

C. Prethreshold Type I ROP

Defined as
- Any ROP in zone I accompanied by plus disease or
- Stage 3 to any extent in zone I or
- Stage 2 or 3 in zone II with plus disease

10. Enumerate tests for macular disorders. (3 marks)

1. Amsler grid
2. Color vision test
3. Photostress recovery test
4. Visual field analysis 10–2
5. Optical coherence tomography

11. Describe the FFA findings in CSCR. (3 marks)

- Fundus fluorescein angiography demonstrates a leak in the arterial phase which increases in intensity and size with time

- The leak is due to dye pooling into the subretinal space
- It is called the expansile dot pattern and more commonly seen
- Another pattern is the smokestack pattern, wherein the dye appears as a dot and then slowly moves up vertically like a rising plume of smoke
- It is due to the convection currents and protein concentration gradient between subretinal fluid and the fluorescein dye
- This unique pattern is however seen only in 10% of the cases

12. Describe the clinical features of solar retinopathy. (3 marks)

Solar retinopathy is classically described as eclipse retinopathy or photoretinitis caused by damage to the photoreceptor complex due to a thermal injury caused by the heat.

Symptoms

Patients typically present around 4 hours after exposure with distorted vision and central scotoma.

Signs

- Visual acuity at presentation may vary from 6/60-6/12 and may improve to 6/12-6/6 after 6 months
- Fundus shows a well yellow spot at the fovea with a gray margin in the perifoveal region
- After a few hours, the yellow spot is replaced by a defect in the fovea with irregular borders
- A positive scotoma is discovered by performing an Amsler test.

13. Enumerate three clinical variants of retinitis pigmentosa. (3 marks)

1. **Inverse retinitis pigmentosa**
   - It is characterized by bony spicule pigmentation seen in the macula area and a decrease in the central vision
2. **Retinitis pigmentosa sine pigmento**
   - It lacks the characteristic pigmentation in the form of bony spicules, but has other features of RP
3. **Sector retinitis pigmentosa**
   - The presence of pigmentation in only one quadrant of the retina, usually inferior

14. Describe the features of a congenital retinal disorder characterized by oculodigital reflex. (3 marks)

- Oculodigital reflex is repetitive or frequent eye rubbing or poking by the child
- It is indicative of low vision
- The congenital retinal disorder characterized by oculodigital reflex is Leber congenital amaurosis
- It is the most common genetic cause of visual impairment characterized by severe rod and cone dysfunction

Clinical Features

Symptoms

There is severe visual impairment and these patients present with delayed visual milestones in the first year of life.

Signs

- Oculodigital reflex: The affected children tend to indent on the globe to induce some visual sensations
- Anterior segment is normal
- Pupils are non-reactive (APD)
- Fundus findings are normal in the initial stages
- Later, arterial attenuation, coloboma-like appearance of the macula, and bone spicule pigmentation in the periphery are seen

15. Describe the ocular features of oculocutaneous albinism. (3 marks)

Oculocutaneous Albinism

- Oculocutaneous albinism is an autosomal recessive
- Ocular albinism is an X-linked recessive and is of two types
- There is decreased or nil production of melanin because of defective tyrosinase activity
- Melanin is conspicuously absent from skin, hair, and uvea

Ocular Features

Iris

- Pink appearing iris due to the extremely thin stroma through which fundal glow is visible
- Transillumination defects in iris
- Severe photophobia

Choroid

The pigment is absent, so underlying sclera and overlying retinal and choroidal vessels are visible clearly.

Retina

- Foveal hypoplasia is partly formed or absent fovea
- As a result, these patients have pendular nystagmus and poor central vision

16. Differentiate between choroidal detachment (CD) and retinal detachment (RD). (3 marks)

Differences in CD and RD

Feature	Choroidal detachment	Retinal detachment
Pathology	The separation between choroid and sclera	The separation between RPE and neurosensory detachment
Color of detachment	Brown	Pale
Color of retinal vessels	Red	Dark red
Shape	Dome-shaped	Depends on the cause and site of the break
Location	Occurs between periphery and equator, not posterior to vortex veins. May extend beyond ora serrata	Occurs between retinal break and the ora serrata (rhegmatogenous) Depends on the site of tractional bands (in t-RD)
IOP	Very low	Depends on the type of retinal detachment RRD: Low Exudative and tractional RD: Normal
B-san ultrasound	Kissing choroidal Dome-shaped immobile masses in the equatorial regionss	Undulating movements of the retina are seen

17. Enumerate three causes for tractional detachment. (3 marks)

1. Proliferative diabetic retinopathy
2. Retinopathy of prematurity
3. Eales' disease

18. Enumerate three causes for exudative retinal detachment. (3 marks)

1. Pregnancy-induced hypertension.
2. Posterior scleritis
3. VKH syndrome

19. Explain the evaluation of an eye with retinoblastoma. (3 marks)

Evaluation of a Case of Retinoblastoma

1. Fundus examination with a dilated pupil of both eyes to note the type, extent, laterality, and the focality of the tumor. Scleral indentation is a must
2. Intraocular pressure assessment to note the anterior chamber extension
3. CT scan of orbit and head to note intracranial extension
4. Ultrasound of the eye to note the size of the tumor. Its characteristics are extensive calcification within the tumor
5. Ultrasonography of the neck and abdomen can also be done
6. Genetic analysis: it is done with biopsy specimens from the enucleated eye and blood for DNA sampling

20. Differentiate between endophytic and exophytic retinoblastomas. (3 marks)

Differentiating Features

Endophytic retinoblastoma	Exophytic retinoblastoma
• Growth of tumor into the vitreous cavity • The tumor looks like a well-circumscribed polypoidal mass of white or pearly pink in color • Fine blood vessels and sometimes a hemorrhage may be present on its surface • In the presence of calcification, it gives the typical 'cottage cheese' appearance • There may be multiple growths projecting into the vitreous	• It grows outwards and separates the retina from the choroid leading to elevation of the retina • On fundus examination, it gives an appearance of exudative retinal detachment • It is a solid detachment and transillumination is negative • Retinal vessels are seen over the surface

21. Explain the salient points of enucleation in retinoblastoma. (3 marks)

Enucleation in Retinoblastoma

- Care should be taken not to damage the globe
- The optic nerve is cut as far as possible by pulling the globe out and using straight scissors
- The globe is inspected for any scleral extension and the optic nerve is measured
- The resected globe is sent for detailed histopathology analysis

22. Describe the superiority of optical coherence tomography angiography (OCTA) over fluorescein angiography. (3 marks)

- Optical coherence tomography angiography is noninvasive and does not require an injection of any drug. So, there are no adverse reactions related to fluorescein like anaphylaxis

- It is much faster than fluorescein angiography which takes at least 15–20 minutes
- OCT-A provides three-dimensional images whereas fluorescein angiography gives two-dimensional images. It provides volumetric angiography images and detailed delineation of the foveal avascular zone
- Segmentation of different layers is not possible in fluorescein angiography which is easily studies using OCT-A
- The use of a montage view using OCT-A provides a wider view with improved resolution

23. Explain the timing and method of screening for ROP. (3 marks)

Timing of Screening

- Gestational age <32 weeks
- Birth weight <1500 g
- The first examination should be done between 6 and 7 weeks postnatal age or 34 weeks post-conceptional age (whichever is earlier)
- Depending on the progression, review screening is performed till the threshold disease is reached and treatment is given. If ROP does not develop, screening is done till 40 weeks of gestation

Procedure of Screening

- The pupils are dilated with tropicamide 0.5% and phenylephrine 1% (diluted drops)
- The entire retina is screened by indirect ophthalmoscopy preferably with a 30D lens
- Scleral indentation is done to view the extreme periphery

24. Enumerate six causes for peripheral retinal neovascularization. (3 marks)

1. Diabetic retinopathy
2. Retinal venous occlusions
3. Retinopathy of prematurity
4. Eales' disease
5. Sickle cell anemia
6. Intermediate uveitis

COMPETENCY

OP 8.5 DESCRIBE AND DISCUSS THE CORRELATIVE ANATOMY, AETIOLOGY, CLINICAL MANIFESTATIONS, DIAGNOSTIC TESTS, IMAGING AND MANAGEMENT OF DISEASES OF THE OPTIC NERVE AND VISUAL PATHWAY

LONG ESSAYS

1. Describe the anatomy of the visual pathway with a neat, labeled diagram and explain the field defects associated with lesions of each portion of the pathway. (5+5 marks)

Anatomy of the Visual Pathway

The visual pathway begins at the retina (Fig. 8.5.1)

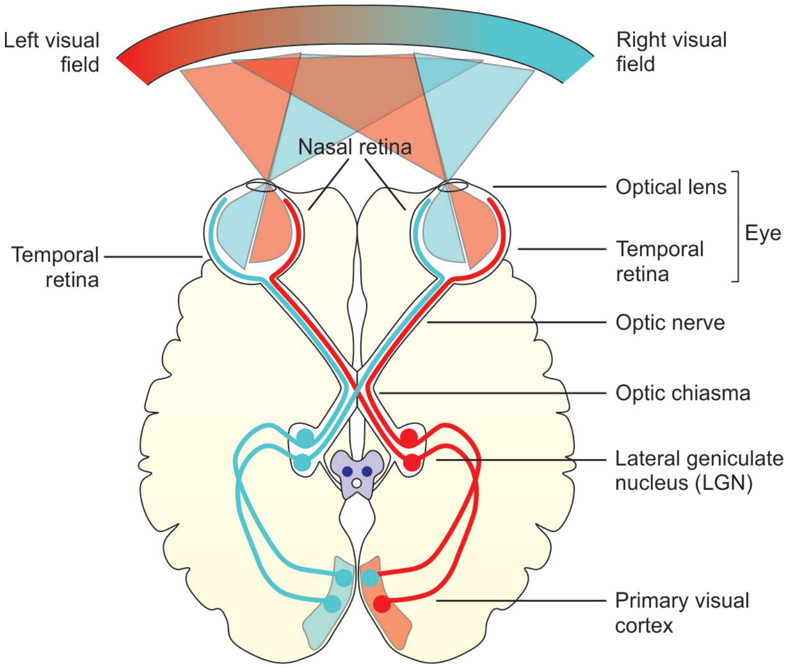
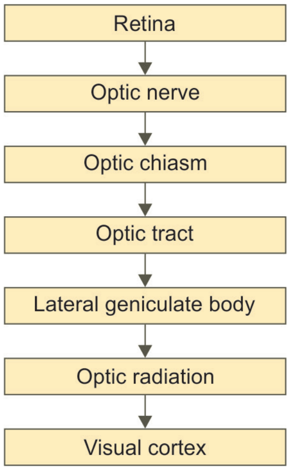

Left visual field
Right visual field
Nasal retina
Optical lens
Eye
Temporal retina
Temporal retina
Optic nerve
Optic chiasma
Lateral geniculate nucleus (LGN)
Primary visual cortex

Fig. 8.5.1: Anatomy of the visual pathway


Retina
Optic nerve
Optic chiasm
Optic tract
Lateral geniculate body
Optic radiation
Visual cortex

1. **Optic nerve**
   - It extends from the lamina cribrosa to the optic chiasm and is about 50 mm in length
2. **Optic chiasma**
   - It is a flattened 'X' shaped structure caused by intersecting fibers from both the optic nerves
3. **Optic tract**
   - These extend from chiasm and fibers terminate at the lateral geniculate body
   - Hereafter the fibers become second-order neurons
4. **Optic radiation**
   - These carry information from ipsilateral temporal and contralateral nasal retinae, and the nerves are getting organized as they travel posteriorly, i.e., nerves carrying close retinal elements travel closely
5. **Visual cortex**
   - It is located on the medial aspect of the occipital lobe, above and below the calcarine fissure
   - The macula is represented by the tip of the lobe
   - Area 17 is the visuosensory area and 18 and 19 form the visuopsychic area

Lesions of the Visual Pathway and Resultant Defects

1. **Lesion at the optic nerve**
   - Visual acuity drops

- Dyschromatopsia
- Afferent pupillary defect
- Field loss: (Fig. 8.5.2)
- Central scotoma: Macular affection
- Altitudinal: Seen in ischemic optic neuropathy
- Complete loss of visual field: Seen in optic atrophy

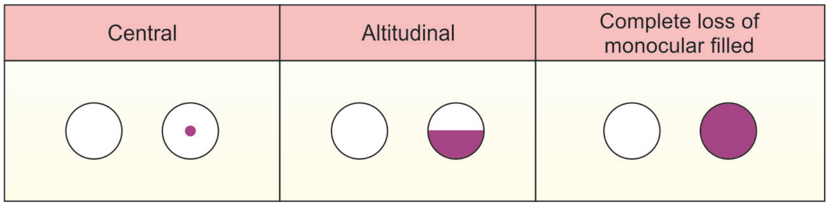


Fig. 8.5.2: Types of field defect in optic nerve lesions

2. **Lesions just anterior to the chiasma**
   - Junctional scotoma characterized by ipsilateral total loss of vision with contralateral incomplete temporal hemianopia (Fig. 8.5.3)
3. **Sagittal lesions at the chiasm**
   - Bitemporal hemianopia, bitemporal hemianopic loss of pupillary reflexes (Fig. 8.5.4)
4. **Lateral lesions at the chiasm**
   - Binasal hemianopia (Fig. 8.5.5), binasal hemianopic loss of pupillary reflexes
5. **Lesions of the optic tract**
   - Incongruous contralateral homonymous hemianopia (Fig. 8.5.6) with contralateral hemianopic pupillary reaction
   - Optic atrophy
6. **Lesions of the lateral geniculate body**
   - Incongruous contralateral homonymous hemianopia (Fig. 8.5.6)
   - Pupil spared, partial optic atrophy
7. **Lesion of the optic radiation**
   - Complete homonymous hemianopia
   - Superior quadrantanopia—temporal lobe lesions (Fig. 8.5.7)
   - Inferior quadrantanopia in parietal lobe lesions (Fig. 8.5.8)
   - No loss of pupil reflex
   - No optic atrophy

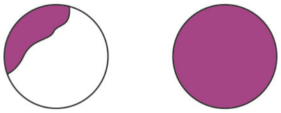


Fig. 8.5.3: Junctional scotoma

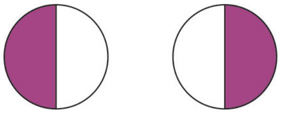


Fig. 8.5.4: Bitemporal hemianopia

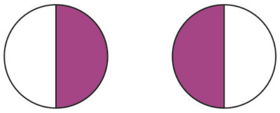


Fig. 8.5.5: Binasal hemianopia

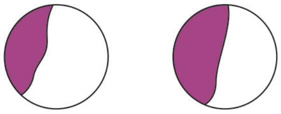


Fig. 8.5.6: Incongruous homonymous hemianopia

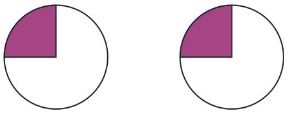


Fig. 8.5.7: Superior quadrantanopia

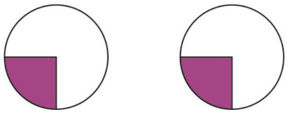


Fig. 8.5.8: Inferior quadrantanopia

8. **Lesions of the visual cortex**
   - Congruous homonymous hemianopia with macula sparing (Fig. 8.5.9)
   - Normal pupillary reactions

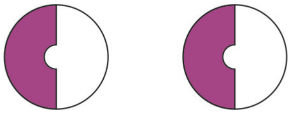


Fig. 8.5.9: Homonymous hemianopia with macula sparing

2. A 45-year-old lady complains of drooping of eyelids and diplopia which is more towards the end of the day, and she is completely asymptomatic after her sleep. Also, she complains of easy fatiguability on exertion.

A. What is the most probable diagnosis from this history? (1 mark)
B. Describe the pathophysiology of this condition. (3 marks)
C. Describe the signs and symptoms of this condition. (3 marks)
D. Describe the investigations and management of this condition. (3 marks)

A. Most Probable Diagnosis

Myasthenia gravis with predominantly ocular manifestations (Ocular myasthenia)

B. Pathophysiology

- It is a potentially fatal autoimmune condition in which the body develops T-cell mediated immunity against acetylcholine receptors in the postsynaptic membrane in the myoneural junction of the skeletal muscles (Fig. 8.5.10)
- Ocular muscles are very early to be affected as they are fast-twitch muscles
- The activated T cells arise from a thymoma
- These produce antibodies against the receptors for acetylcholine in the postsynaptic membrane
- As a result, the acetylcholine that is released gets destroyed in the synaptic cleft without performing its action
- In the postsynaptic membrane, there is no activation of sodium channels and the formation of potential
- As a result, the muscles cannot contract

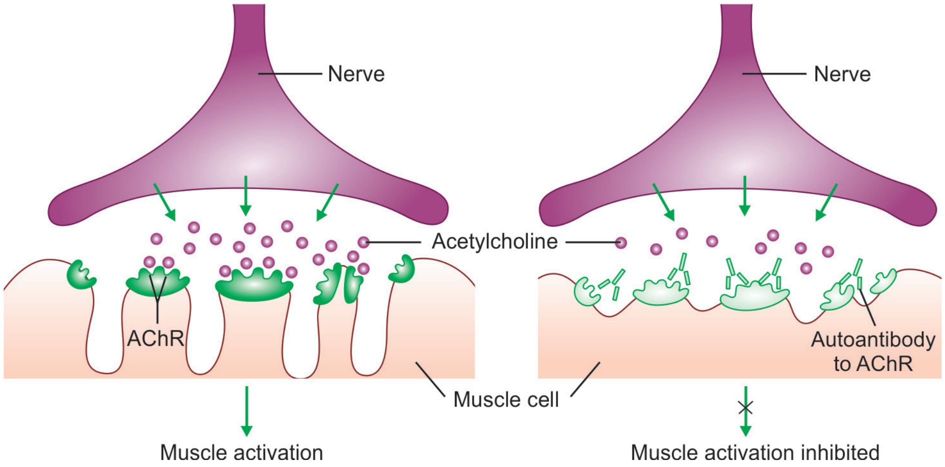


Fig. 8.5.10: Pathogenesis of myasthenia gravis

C. Signs and Symptoms

Structure	Symptoms and signs	Clinical tests
Eyelids • The first to be affected as eyelid muscles are fast-twitch fibers	Ptosis, unilateral or bilateral, asymmetric, variable with time	Eyelid fatiguability test • Increase in ptosis after prolonged upgaze Cogan's lid twitch • The patient is instructed to look down continuously for 15 seconds and then asked to fixate on a point in primary gaze. The upper lid shoots up briefly and then comes back to its original position Positive ice test • Improvement of ptosis after placing ice on the lid for 2 minutes
Extraocular movements	Diplopia: Can be horizontal or vertical, asymmetric, variable	Does not fit into a recognizable pattern Improvement after rest and edrophonium

D. Investigation and Treatment

Investigations

1. **Edrophonium test**
   - It is a short-acting anticholinesterase agent that increases the availability of acetylcholine at the neuromuscular junction
   - Dose: 2 mg intravenous dose after 0.3 mg atropine IV
   - Followed by 8 g after 60 seconds once hypersensitivity is ruled out
   - Subjective improvement in ptosis or diplopia is recorded with the help of a Hess screen
2. **Chest X-ray**
   - To rule out thymoma

Treatment

- Neostigmine
- Pyridostigmine
- Plasmapheresis
- Intravenous immunoglobulins (IVIG)
- Systemic steroids
- Thymectomy (only in generalized MG)

3. Enumerate the parts of the optic nerve and describe their characteristics. Describe various tests for optic nerve function. (5+5 marks)

Parts of the Optic Nerve

It has four parts (Fig. 8.5.11)

1. **Intraocular part (1 mm)**
   - Has four parts: Surface, prelaminar, laminar and post-laminar
   - Supplied by the short posterior ciliary arteries and central retinal artery which form a circle of Zinn–Haller

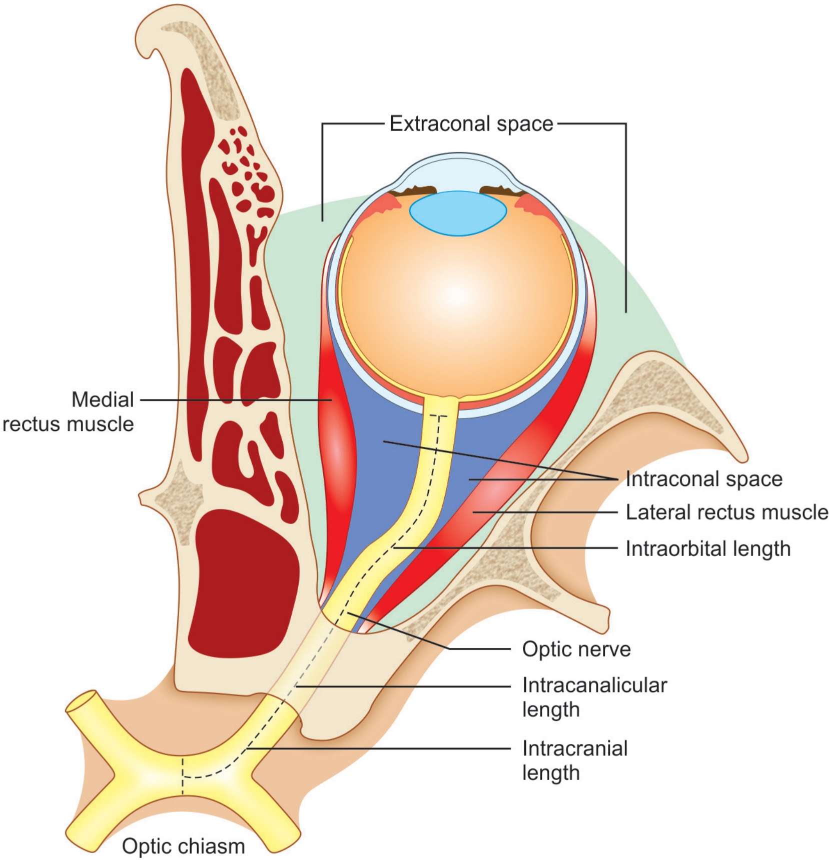

Extraconal space
Medial rectus muscle
Intraconal space
Lateral rectus muscle
Intraorbital length
Optic nerve
Intracanalicular length
Intracranial length
Optic chiasm

Fig. 8.5.11: Parts of the optic nerve

2. **Intraorbital part (25 mm)**
   - Sinuous, to allow movements of the eyeball, a certain amount of proptosis is tolerated due to this
   - Closely associated with the annulus of Zinn where extraocular muscles originate, more so to medial rectus
   - Reason for pain during eye movements in optic neuritis
   - It is supplied by branches of the ophthalmic artery
3. **Intracanalicular part (4–10 mm)**
   - Most susceptible to damage by traumatic optic neuropathy due to close space
   - Sphenoid and posterior ethmoidal sinuses lie medial to it and are separated by a thin bony lamina
   - As a result, optic neuritis frequently follows sinusitis
   - The ophthalmic artery lies inferotemporal
4. **Intracranial part (10 mm)**
   - Present above the cavernous sinus
   - Supplied by the anterior cerebral artery

Tests for Optic Nerve Function

1. **Visual acuity**
   - Affected most of the time

- May range from perception of light negative to 6/6
- Patients with optic perineuritis (inflammation of sheath of the optic nerve) tend to have a better central vision. It can be associated with syphilis, giant cell arteritis, and granulomatosis with polyangiitis (Wegener's granulomatosis)

2. **Color vision abnormalities or dyschromatopsia**
   - Always present, mainly in the form of red-green disturbances (cf blue-yellow in macular disorders)
   - Can be tested by Farnsworth Munsell 100 hue test, Holmgren's test, Farnsworth D15 hue discrimination test, and Naegels anomaloscope
   - Ishihara's chart also can be used, but it is mainly designed to recognize congenital color blindness
3. **Pupillary abnormality**
   - The lesions of the optic nerve always give rise to a Marcus Gunn pupil or a relative afferent pupillary defect and there is no anisocoria
   - When the lesion is bilaterally symmetric, there is no RAPD
4. **Visual field**
   - Optic nerve lesions can cause a variety of visual field defects
   - Sometimes it may not be possible to have classical field defects in some disorders
   - However, the classical field defects are listed below
     1. Centrocecal and central scotoma in toxic amblyopia, LHON, Nutritional optic neuropathy
     2. Altitudinal (superior or inferior) scotoma in ischemic optic neuropathy
     3. A combination of central, cenroacecal, paracentral, and arcuate scotomas can be seen in optic neuritis
5. **Disc appearance**
   - The disc may be normal, pale, or edematous
   - Normal looking disc
     - Retrobulbar neuritis (optic neuritis)
     - Traumatic optic neuropathy (initial stages),
     - Posterior ischemic optic neuropathy (initial stages)
     - Pituitary apoplexy (initial stages)
   - Disc edema
     - Optic neuritis—can present as papillitis
     - Optic disc edema with macular star (ODEMS) or neuroretinitis
     - Papillophlebitis
     - Ischemic optic neuropathy
     - Initial stages of compressive optic neuropathy and Leber's hereditary optic neuropathy
   - Pale disc
     - Any optic neuropathy over time develops optic atrophy and the disc appears pale. But these conditions develop pallor without developing edema
     - Toxic and nutritional optic neuropathy (temporal pallor)
     - Hereditary optic neuropathy (diffuse pallor)

- Compressive optic neuropathy (from the optic nerve to lateral geniculate body)
- Chiasmal compression causes bow-tie optic atrophy

6. **Visually evoked potential**
   - It is an objective electrophysiological test that tests the integrity of the afferent visual pathway
   - It is a noninvasive test, where surface electrodes are placed on the scalp in the occipital area and visual stimuli are shown to the individual
   - The recordings are amplified and demonstrated on the computer screen
   - The recordings are unaffected by medial opacities, and thus can be used to prognosticate visual outcomes or understand optic nerve status
   - It is nonspecific and cannot localize the lesion
   - The normal VEP has an initial negative wave at 75 ms called the N75, an initial positive wave at 100 ms called the p100, and a second negative wave at 135 ms
   - The amplitude of the p100 is considered normal if the value is between 10 and 25 microvolt

4. A 33-year-old woman presented with severe headaches and vomiting to the ER. On examination, both the optic discs are swollen. The resident made a diagnosis of papilledema

A. Define papilledema. (1 mark)

B. Enumerate the differences between papilledema and papillitis. (4 marks)

C. Give one cause for bilateral papillitis and one cause for unilateral papilledema. (2 marks)

D. Explain the disc findings in papilledema. (3 marks)

A. Definition of Papilledema

Usually bilateral, non-inflammatory, passive swelling of the optic nerve head following an increase in the intracranial pressure.

B. Difference between Papilledema and Papillitis

Feature	Papilledema	Papillitis
Laterality	Bilateral usually	Unilateral usually
Symptoms	Transient loss of vision Painless	Sudden diminution of vision Pain on extraocular movements
Pupillary reactions	Normal	RAPD
Media	Clear	Posterior vitreous haze
Disc elevation	2–6D	Does not exceed 2–3D
Venous engorgement, hemorrhages	More frequent	Less common

C. Cause of Bilateral Papillitis and Unilateral Papilledema

Cause for Bilateral Papillitis

Cat scratch disease.

Cause for Unilateral Papilledema

Optic atrophy in one eye and increase in intracranial pressure can cause unilateral papilledema (Foster Kennedy syndrome) due to tumor of the olfactory grove.

D. Disc Findings in Papilledema

The fundus findings in papilledema include vascular and mechanical signs

Vascular signs	Mechanical signs
• Venous congestion of arcuate and peripapillary vessels. • Papillary and retinal peripapillary hemorrhages. • Nerve fiber layer infarcts (cotton-wool spots). • Hyperemia of the optic nerve head.	• The blurring of the optic disc margins (superonasal and inferonasal are the first to be involved) • Filled optic disc cup • Elevation of the surface of the disc • Edema of the peripapillary nerve fiber layer • Paton's lines: Circumpapillary gray folds in the nerve fiber layer

5. A 28-year-old Caucasian woman presents with rapid loss of vision in her left eye noticed since the last 6 hours. It is associated with painful eye movements. On examination, visual acuity is 6/6, N6 in the right eye, and Perception of light positive in the left eye, with RAPD of the left eye. Fundus examination is normal in both eyes.

A. State the most probable diagnosis and cause in this woman? (2 marks)
B. Enumerate 4 other causes of the same condition. (2 marks)
C. Describe the management of this patient. (2 marks)
D. Explain the results of ONTT. (2 marks)
E. Enumerate 2 other causes of optic neuropathy characterized by a normal-looking optic disc. (2 marks)

A. Most Probable Diagnosis

- Right-sided optic neuritis, presenting as retrobulbar neuritis
- The most common cause for the same in the Caucasian race presenting in women of the third decade is multiple sclerosis

B. Other Causes for Optic Neuritis

1. Idiopathic—many a time
2. Viral infections like measles, chickenpox, mumps, herpes zoster
3. Autoimmune disorders like SLE, sarcoidosis
4. Local infections like orbital cellulitis, meningitis, sinusitis

C. Management

- An MRI brain with optic nerve imaging, preferably FLAIR sequence is performed to look for plaques suggestive of multiple sclerosis. CSF analysis is performed to rule out NMo antibodies
- Management includes the use of intravenous methylprednisolone 1g in four divided doses for three days
- It is followed by oral prednisolone 1 mg/kg body weight given once a day for 13 days followed by a short taper over 3 days
- The treatment is associated with faster recovery of visual acuity and a lower rate of recurrences

D. Results of the Optic Neuritis Treatment Trial (ONTT)

- Optic neuritis treatment trial was a multicentric study conducted in North America and included cases of typical optic neuritis: aged 18–46 years with acute onset of painful diminution of vision. The pain was seen in most patients as a presenting symptom
- The subjects were divided into three arms: One control group which received oral placebo, one arm with oral prednisolone, and one arm with 3 days of 1g/day intravenous methylprednisolone followed by 13 days of 1g/kg of prednisolone and three days of short taper
- The third group was found to have the fastest visual recovery; however, final visual acuity was unaffected
- The group with oral steroids was found to have higher recurrences and thus is not indicated
- ONTT also helped in prognosticating the development of clinical multiple sclerosis as 50% in patients with one episode of optic neuritis regardless of MRI finding and 72% in patients with one or more plaques seen in MRI. Patients with normal MRI had a 25% chance of development of multiple sclerosis

E. Causes of Optic Neuropathy with Normal-appearing Disc

The optic disc looks normal in the initial stages of the following conditions.

1. Posterior ischemic optic neuropathy
2. Traumatic optic neuropathy

6. A 66-year-old diabetic presents with sudden painless diminution of vision in his right eye. On examination, visual acuity in the right eye is 3/60 and in the left eye, it is 6/6. There is a RAPD, and the disc shows sectoral edema.

A. What is the most probable diagnosis in this case? (1 mark)

B. What is the probable finding in the optic disc of the fellow eye? (1 mark)

C. Differentiate between the two main types of this condition. (5 marks)

D. Explain the anatomical basis for a visual field that is seen in this condition. (3 marks)

A. Most Probable Diagnosis

Right-sided non-arteritic anterior ischemic neuropathy (NAION).

B. Probable Finding in the Optic Disk of the Fellow Eye

The other eye usually has a small and crowded disc.

C. Differences between Arteritic and Non-arteritic Optic Neuropathy

Clinical feature	Arteritic	Non-arteritic
Age of presentation	6th or 7th decade, F>M	5th or 6th decade, M=F
Underlying etiology	Occlusive arteritis due to autoimmune pathology	Remains unknown

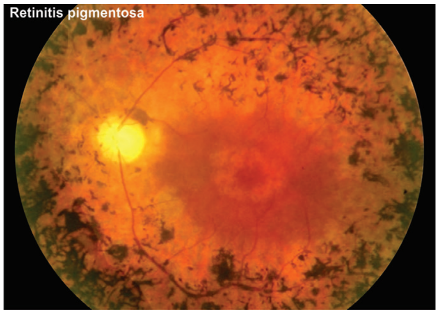
Contd.

Clinical feature	Arteritic	Non-arteritic
Associated conditions	Giant cell arteritis	Uncontrolled attack of angle-closure glaucoma Diabetes, hypertension, massive loss of blood, perioperative loss of blood, and severe anemia. Drugs like sildenafil
Visual acuity	Transient loss of vision may preclude sudden visual loss It May also be associated with CRAO Visual loss is usually marked, the other eye involvement is as high as 95% Diplopia due to ischemia of extraocular muscles	Visual acuity is not as marked as AAION, the other eye involvement is around 30%
Other features	Jaw claudication, scalp tenderness, severe one-sided headache Polymyalgia rheumatica	Painless Disc at risk or a crowded disc, where the disc is small, and the axons are packed
Disc appearance	Pallid edema associated with cotton wool spots and splinter hemorrhages	Sectoral edema, hyperemia present The other eye has a small cup
Fundus fluorescein angiography	Delayed choroidal filling and disc filling	Disc filling delay only
Laboratory parameters	Raised ESR, CRP Temporal artery biopsy confirms the diagnosis	Nonspecific
Treatment	High dose corticosteroids	No proven treatment Identification and control of systemic features Oral prednisolone has been proven to be useful in around 60–70% showing visual improvement Levodopa-carbidopa combination for 3 weeks and then double the dose for a month Intravitreal erythropoietin has also been tried with a short-term success
Prognosis	Poor	Recovery is seen in around 50% of individuals

D. Anatomical Basis for Visual Field Defect

- The visual field defect that is seen in anterior ischemic optic neuropathy is altitudinal scotoma, which is either superior or inferior
- Anterior ischemic optic neuropathy affects the prelaminar region of the intraocular optic nerve
- This region is mainly supplied by the arterial arcade formed by short posterior arteries which divide into two main trunks-superior and inferior before giving rise to smaller branches
- In AION, any one trunk is affected
- As a result, an altitudinal visual field defect is seen

7. A 54-year-old man comes to the government ophthalmic outpatient department for certification of blindness for monetary compensation for blindness. On examination, you find that the visual acuity is PL+, PR accurate in both eyes. Pupils are sluggishly reactive, and the fundus shows the following picture.



A. What is the most probable diagnosis? What is the type of optic atrophy in this patient? (2 marks)

B. Explain Kestenbaum number and Kestenbaum sign. (2 marks)

C. Explain the morphological classification of optic atrophy with their clinical signs and one cause each. (4 marks)

D. Name 2 optic neuropathies that present initially with optic disc pallor. (2 marks)

A. Most Probable Diagnosis

Retinitis pigmentosa and the type of optic atrophy is consecutive optic atrophy.

B. Kestenbaum Number and Sign

Kestenbaum Number

- Kestenbaum suggested that optic atrophy is due to a decrease in the vasculature of the disc
- Normal Kestenbaum number is 10 capillaries that can be counted on the disc surface on slit lamp biomicroscopy with high magnification

Kestenbaum Sign

- When the number of small capillaries on the surface of the disc is less than 6 and is considered optic atrophy
- >12 is considered hyperemia

C. Morphological Classification of Optic Atrophy

Type	Causes	Clinical Signs
Primary optic atrophy	• It results due to a primary optic nerve disease *per se* • Causes include ▪ Demyelination ▪ Gliomas ▪ Tumors compressing anterior visual pathway ▪ Traumatic avulsion ▪ Toxic amblyopia ▪ LHON	• The disc is chalky white • Sharp clear margins • Retinal vessels are normal in caliber • Cup is normal in size, deep
Secondary/post-papilledema optic atrophy	• It follows inflammation of the optic nerve due to infections or papilledema	• The disc is greyish white • Margins are fuzzy due to gliosis • Vessels have sheathing

Contd.

Type	Causes	Clinical Signs
Consecutive optic atrophy	• It is associated with a degenerative process involving the choroid and retina. • Causes include ▪ Retinitis pigmentosa ▪ Pathological myopia	• The waxy yellow color of the disc • Vessels are attenuated • Margins are fuzzy
Glaucomatous optic atrophy	• It occurs due to chronic uncontrolled glaucoma either due to primary or secondary causes • It may follow an open-angle or closed-angle glaucoma	• Disc is pale • Cup is deep and large • Vessels are nasalized • Loss of retinal nerve layer can be seen • Laminar dot sign: Pores in the lamina cribrosa are seen due to thinning

D. Optic Neuropathies Presenting Initially with Optic Disc Pallor

1. Toxic and nutritional optic neuropathy (temporal pallor)
2. Hereditary optic neuropathy (diffuse pallor)
3. Compressive optic neuropathy (from the optic nerve to lateral geniculate body)

8. Enumerate the types of retrochiasmal lesions and their common features. Explain the salient features of each type that help in localizing the site of the lesion. (4+6 marks)

Retrochiasmal Lesions

1. Optic tract
2. Lateral geniculate body
3. Optic radiation
4. Occipital cortex
5. Tip of the occipital cortex

Common Causes

1. Vascular causes
2. Demyelination
3. Tumors
4. Trauma

Common Features

- Visual field defects are always homonymous (one side of the visual field) and involve the contralateral visual space, e.g. left-sided lesions cause right-sided homonymous hemianopia
- Congruity increases with the posterior location of the lesion. Congruity means how similar the filed defects are to each other if superimposed
- Associated neurological features and congruity help in localizing lesions
- No pupillary abnormality or optic atrophy in lesions beyond LGB

Salient Features of Each Type of Retrochiasmal Lesion

Type	Relevant anatomy	Clinical features
Optic tract lesions	• Each optic tract consists of ipsilateral temporal fibers and contralateral nasal fibers • They are near the cerebral peduncles before they terminate in the lateral geniculate bodies • The nerve fibers containing corresponding retinal elements are not aligned and thus the field defects tend to be incongruous	• Ocular features include Wernicke's hemianopic pupil and bow-tie optic atrophy. • Visual field defects include contralateral hemianopia • Wernicke's hemianopic pupil • Contralateral paraplegia • The optic atrophy in optic tract lesions is unique • On the ipsilateral side, the disc shows pallor of the superior and inferior poles due to the affection of the temporal arcuate fibers • On the contralateral side, there is bow-tie atrophy due to compression of the nasal macular and nasal retinal fibers. As a result, there is pallor on nasal and temporal aspects of the disc
Lateral geniculate body lesions	• Each LGB consists of ipsilateral temporal fibers and contralateral nasal fibers	• Like optic tract lesions, except that the pupil is spared and there is no optic atrophy.
Optic radiation lesions	• The optic radiation extends from the lateral geniculate body to the occipital cortex • The lower retinal fibers (Baum's loop) pass through the parietal cortex and the upper retinal fibers (Meyer's loop) pass through the temporal cortex	• Lesions of the Baum's loop cause contralateral homonymous inferior quadrantanopia. Associated features include acalculia, agraphia, left-right disorientation, and asymmetric optokinetic nystagmus • Lesions of Mayer's loop cause paroxysmal gustatory and olfactory hallucinations (uncinate fits), contralateral hemiparesis, seizures, and dysphasia if the dominant lobe is involved • Optokinetic nystagmus is absent in the direction of the lesion
Occipital cortex	• The visual cortex is formed by the medial aspect of the occipital lobe, above and below the calcarine fissure • The primary visual cortex is formed by area 17 and areas 18 and 19 form the secondary visual cortex	• Contralateral congruous homonymous hemianopia with macular sparing • Homonymous hemianopic scotoma is seen in the involvement of the occipital cortex

9. A 14-year-old boy with neuroblastoma is brought by his parents for a refractive check-up. On examination, visual acuity is 6/6, N6 in both eyes after providing a myopic correction. However, it is noticed that there is mild ptosis, and the pupil of the left eye looks smaller. On the swinging flashlight test, the left pupil is 2 mm smaller than the right pupil.

A. What is the most probable diagnosis? (1 mark)

B. Elucidate the oculosympathetic pathway. (3 marks)

C. Enumerate the causes of lesions at different levels. (3 marks)

D. Create an algorithm of pharmacological tests to localize the site of lesion in this condition. (3 marks)

A. Most Probable Diagnosis

Left-sided Horner's syndrome.

B. Oculosympathetic Pathway (Fig. 8.5.12)

Posterior hypothalamus
↓
Descends along the brainstem
↓
Ciliospinal center of Budge, between C8 and T2
↓
Preganglionic fibers traverse along apical pleura and synapse at the superior cervical ganglion
↓
Postganglionic fibers again enter the cranial cavity by ascending close to internal carotid and travel within the substance of the cavernous sinus
↓
Ophthalmic division of trigeminal nerve
↓
Ciliary ganglion (but does not relay here)
↓
Long ciliary nerves
↓
Dilator pupillae, Muller's muscle

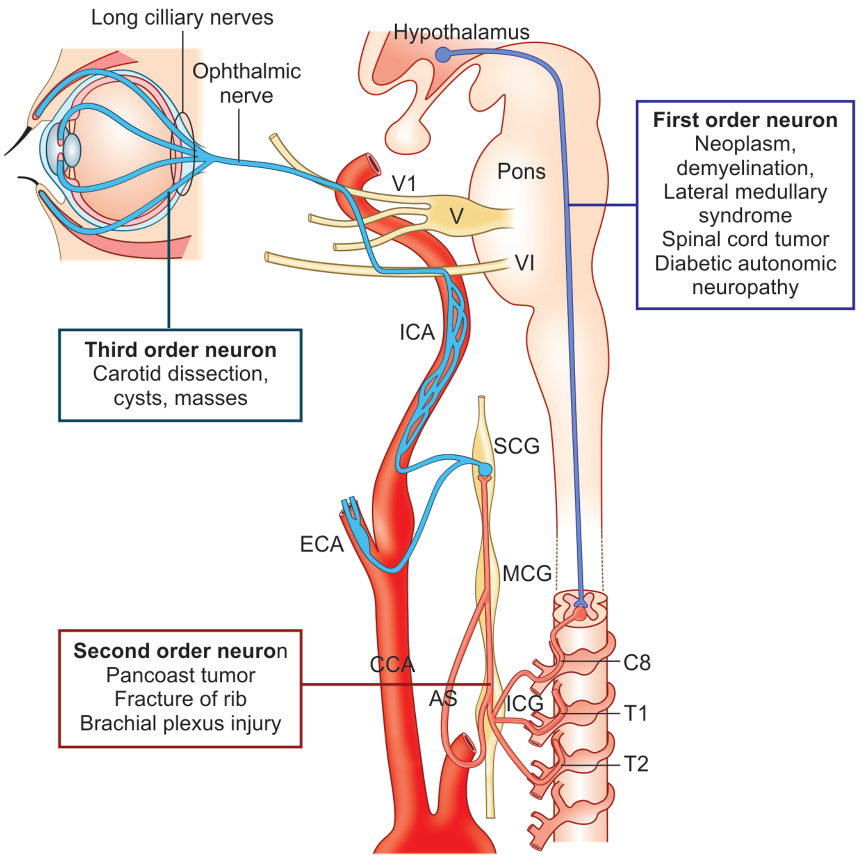

Long cilliary nerves
Ophthalmic nerve
Hypothalamus
Pons
V1
V
VI
ICA
SCG
ECA
MCG
CCA
AS
ICG
C8
T1
T2
First order neuron
Neoplasm, demyelination, Lateral medullary syndrome
Spinal cord tumor
Diabetic autonomic neuropathy
Third order neuron
Carotid dissection, cysts, masses
Second order neuron
Pancoast tumor
Fracture of rib
Brachial plexus injury

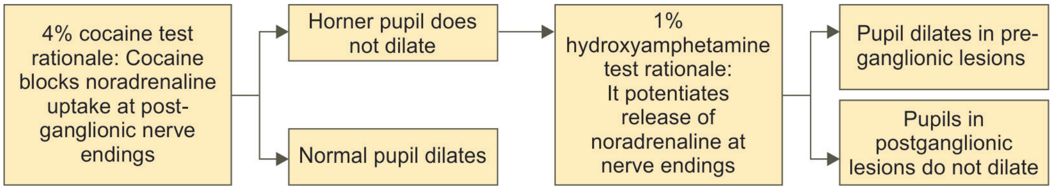
Fig. 8.5.12: Oculosympathetic pathway and causes for Horner's syndrome

C. Causes of Lesions at Different Levels

Central	Preganglionic	Post ganglionic
• Neoplasms, demyelination	• Pancoast tumor	• Cluster headache
• Lateral medullary syndrome	• Carotid aneurysm	• Cavernous sinus thrombosis
• Spinal cord tumor	• Neck lesions like tumors, cysts	• Nasopharyngeal tumor
• Diabetic autonomic neuropathy		• Otitis media

D. Pharmacological Tests for Horner's Syndrome

- It is mainly clinical diagnosis, and drug tests help in localizing the lesion, whether preganglionic or posts ganglionic


4% cocaine test rationale: Cocaine blocks noradrenaline uptake at post-ganglionic nerve endings
Horner pupil does not dilate
Normal pupil dilates
1% hydroxyamphetamine test rationale: It potentiates release of noradrenaline at nerve endings
Pupil dilates in pre-ganglionic lesions
Pupils in postganglionic lesions do not dilate

- Other tests to distinguish pre- and postganglionic lesions are: 1% phenylephrine: in postganglionic lesions pupil dilates due to denervation supersensitivity whereas, in preganglionic lesions, the pupil does not dilate

10. Describe anisocoria with an example and list its causes. Describe the work-up of a case of anisocoria. (5+5 marks)

- Anisocoria is defined as the unequal size of pupils.
- A difference of 2 mm between the 2 pupils is considered anisocoria
- It occurs because of an efferent pathway defect.

Example of Efferent Pupillary Defect (EPD) (Fig. 8.5.13)

Light on the right eye	Right eye pupil 6 mm	Left eye pupil 3 mm
Light on the left eye	Right eye pupil 6 mm	Left eye pupil 3 mm

Causes

- Causes of EPD are lesions of the superior colliculus, third nerve palsy, damage to the ciliary ganglion, and short ciliary nerves, damage to sphincter pupillae, and the use of mydriatics
- Physiological anisocoria is a difference of 1 mm between the two pupils which is equal in bright and dim illumination
- If anisocoria worsens in dark, then the smaller pupil is at fault. If anisocoria worsens in bright light then, the larger pupil is at fault. A good slit-lamp examination may give many cues regarding the structure of the pupil and iris

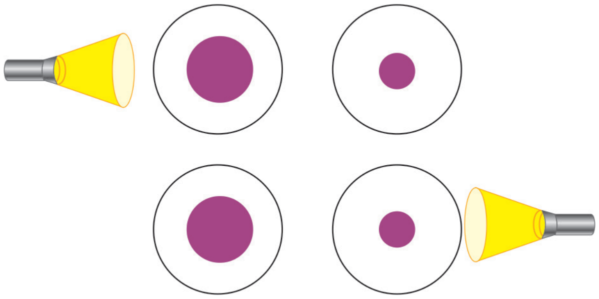
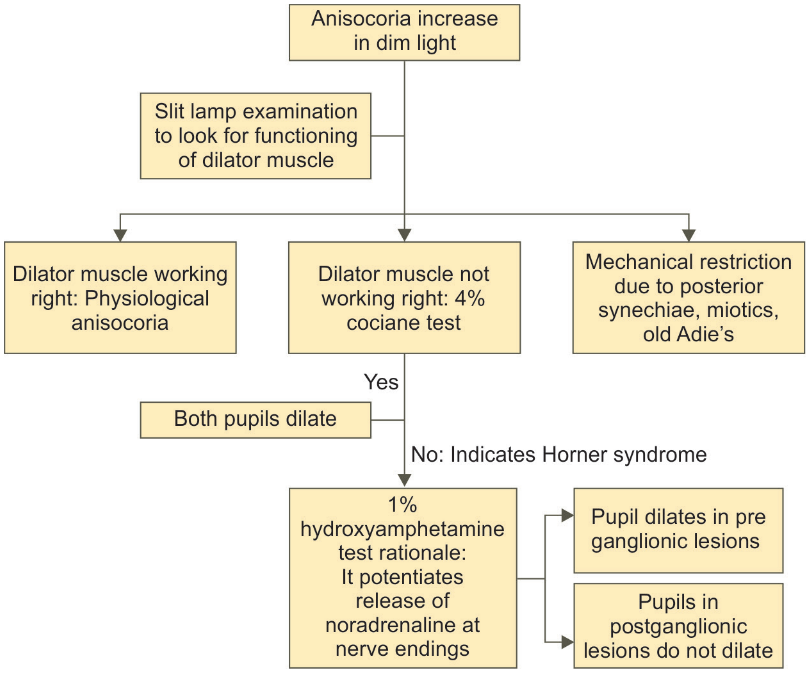


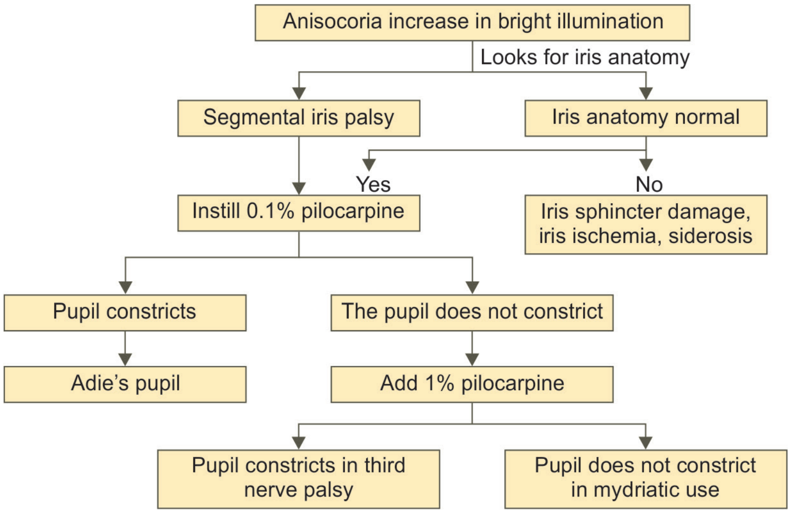
Fig. 8.5.13: Efferent pupillary defect

Causes for anisocoria worsening in dark (small pupil abnormal)	Causes for anisocoria worsening in bright light (Large pupil abnormal)
• Physiological • Horner's syndrome	• Adie's pupil • Third nerve palsy • Use of mydriatic

Work-up of Anisocoria

- The principle behind the work-up for anisocoria is that if anisocoria increases in dark illumination, then the smaller pupil is at fault
- If the anisocoria increases in bright light, then the bigger pupil is at fault
- Symmetric efferent pupillary defects (rare) do not cause anisocoria


Anisocoria increase in dim light
Slit lamp examination to look for functioning of dilator muscle
Dilator muscle working right: Physiological anisocoria
Dilator muscle not working right: 4% cociane test
Mechanical restriction due to posterior synechiae, miotics, old Adie's
Yes
Both pupils dilate
No: Indicates Horner syndrome
1% hydroxyamphetamine test rationale: It potentiates release of noradrenaline at nerve endings
Pupil dilates in pre ganglionic lesions
Pupils in postganglionic lesions do not dilate


Anisocoria increase in bright illumination
Looks for iris anatomy
Segmental iris palsy
Iris anatomy normal
Yes
No
Instill 0.1% pilocarpine
Iris sphincter damage, iris ischemia, siderosis
Pupil constricts
The pupil does not constrict
Adie's pupil
Add 1% pilocarpine
Pupil constricts in third nerve palsy
Pupil does not constrict in mydriatic use

11. A 24-year-old woman presents with an attack of unilateral severe headache associated with nausea and vomiting. Also, she has severe photophobia and prefers to stay in a dark room during the attack.

A. What is the most probable diagnosis? (1 mark)
B. Enumerate three common triggers for this kind of headache. (3 marks)
C. Classify this condition based on its clinical presentation. (3 marks)
D. Explain the management of this condition. (3 marks)

A. Most Probable Diagnosis

Common migraine.

B. Three Common Triggers

1. Menstruation
2. Fasting
3. Lack of sleep

C. Classification of Migraine

• Common migraine or migraine without aura	• Nausea, vomiting, no aura, headache
• Classical migraine (headache with aura)	• Aura followed by headache with or without neurological symptoms
• Acephalic migraine or migraine sine migraine	• Classical migraine features without headache • May have focal neurological disturbances, more common in 40–70 years of age
• Ophthalmoplegic migraine	• More common in the pediatric age group • Migraine headache associated with paralysis of one or more extraocular muscles (usually III nerve)
• Retinal migraine	• Migraine with visual loss
• Basilar artery migraine	• Mimics vertebra-arterial basilar insufficiency with a bilateral blurring of vision, diplopia, vertigo, gait disturbances, visual hallucinations, and dysarthria

D. Management of Migraine

- Treatment of migraines is a step-wise approach. Correction of underlying refractive error and phorias should also be done
- Avoiding stressors and triggers for migraines is the first step. Ingestion of NSAID-antiemetic combination immediately at the onset of headache helps in mild cases
- Lifestyle changes like practicing good sleeping habits, avoidance of alcohol, coffee, and other triggers, practicing yoga must be advocated
- The first line of therapy is acetaminophen/aspirin + metoclopramide
- If the initial therapy fails, the following drugs can be used to terminate an attack
  - 2 mg ergotamine oral (contraindicated in elderly and cardiovascular diseases, peripheral vascular diseases)
  - 6 mg subcutaneous sumatriptan (not to be used in conjunction with ergotamine)
  - 1 mg ergotamine + 10 mg caffeine
  - 4 mg dihydroergotamine

  - Oral 25 mg sumatriptan
  - Butorphanol nasal spray
- Prophylaxis is indicated when there are more than 2 attacks per month or associated neurological features in the form of oral propranolol. Since this patient does not need prophylaxis, propranolol is not indicated

SHORT ESSAYS

1. Describe the staging of papilledema (5 marks)

Hugh and Jackling's staging of papilledema

A. Early/Incipient Stage

- Obscuration of disc margins (superonasal and inferonasal, and finally temporal), absence of spontaneous venous pulsations
- Mild hyperemia of the disc, peripapillary splinter hemorrhages

B. Established Stage

- Obvious elevation of the disc margins
- Filled cup and blurred margins
- Tortuous and engorged veins, hemorrhages
- Paton's lines: circumpapillary gray folds in the nerve fiber layer, cotton wool spots, hard exudates around macula

C. Chronic Papilledema

- Visual acuity is reduced in this stage onwards
- No hemorrhages or cotton wool spots
- Disc appears like dome of champagne cork with crystalline drusen like deposition on the surface: Corpora amylacea
- Cup remains obliterated

D. Atrophic Papilledema

- Visual acuity is severely impaired.
- Fundus appearance resembles postneuritic optic atrophy
- Perivascular sheathing, obscured cup, pale disc, pigmentary changes at macula

2. List the contributions of Dr. Sohan Singh Hayreh to Neuro-ophthalmology. (5 marks)

- The name of Dr. Sohan Singh Hayreh is etched in the history of neuro-ophthalmology with Golden letters due to his contributions to the understanding of various optic nerve disorders
- He did exhaustive research in the anatomy and vasculature of the eye, optic nerve, and orbit
- His contributions are:
  1. He laid down the basis for understanding the pathogenesis of papilledema as axoplasmic stasis
  2. He made revelations regarding the two variants of central retinal venous occlusions
  3. He found that posterior drainage of the vitreous had no result on papilledema

4. He laid down a foundation for understanding the pathogenesis of NAION and glaucomatous optic neuropathy by understanding the circulation of the optic nerve head was through short posterior ciliary vessels
5. He introduced the vasogenic concept of glaucomatous optic nerve damage
6. He described the angiographic pattern of choroidal melanomas and radiation retinopathy
7. He gave strong evidence of the pattern of choroidal circulation and the structure of posterior ciliary arteries. He explained their segmental distribution through many *in vivo* studies. Also, he proved the lobular pattern of the choriocapillaris with arterioles being at the center and venules at the periphery
8. He discovered the presence of water-shed zones between the choroidal vasculature and optic nerve head circulation. This helped in understanding various pathologies of the same
9. He proved that there are no anastomoses between the choroidal vessels
10. He discovered a new clinical entity called optic disc vasculitis and explained management for the same
11. He laid down diagnostic criteria for giant cell arteritis and its ophthalmic manifestations
12. He is a pioneer of central retinal artery occlusions, central retinal venous occlusions, and ischemic optic neuropathies

3. Explain the arrangement of nerve fibers at the optic disc. Justify temporal pallor in toxic amblyopia. (3+2 marks)

Arrangement of Nerve Fibers at the Optic Disc (Fig. 8.5.14)

Nasal Fibers (Radially Arranged)

They represent the temporal field which is the largest of all. So, the nerve fibers are thicker .

Temporal Fibers (Arch around Macular Fibers)

They represent the nasal visual field.

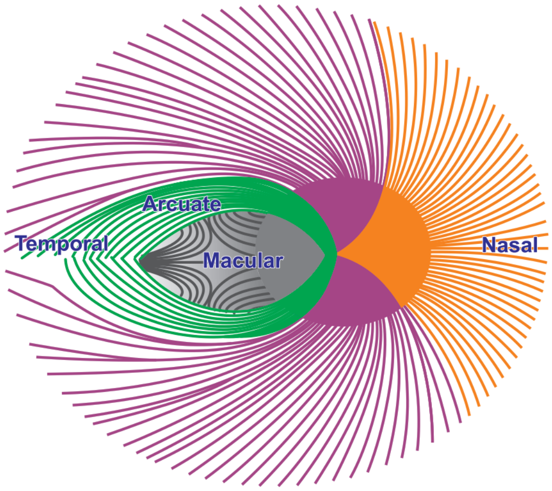


Fig. 8.5.14: Arrangement of nerve fibers in the retina

Macular Fibers (Macula to Optic Disc—Papillomacular Bundle)

These are the thinnest fibers and highly dependent on ATP and have a greater number of mitochondria.

Cause for Temporal Pallor in Toxic Amblyopia

- The papillomacular bundle is a highly active portion of the optic nerve and is dependent on a high quantity of ATP and thus, needs a constant supply of energy
- In toxic amblyopia, there is a toxic agent that causes depletion of ATP or destruction of mitochondria. As a result, there is dysfunction of the papillomacular bundle. It enters the optic disc from the temporal aspect
- Following neuropathy involving these fibers, there is resultant atrophy causing temporal pallor

4. Draw a neat and labeled diagram of the orbital apex and explain its clinical implications. (5 marks)

Diagram of the Orbital Apex

See Figure 8.5.15.

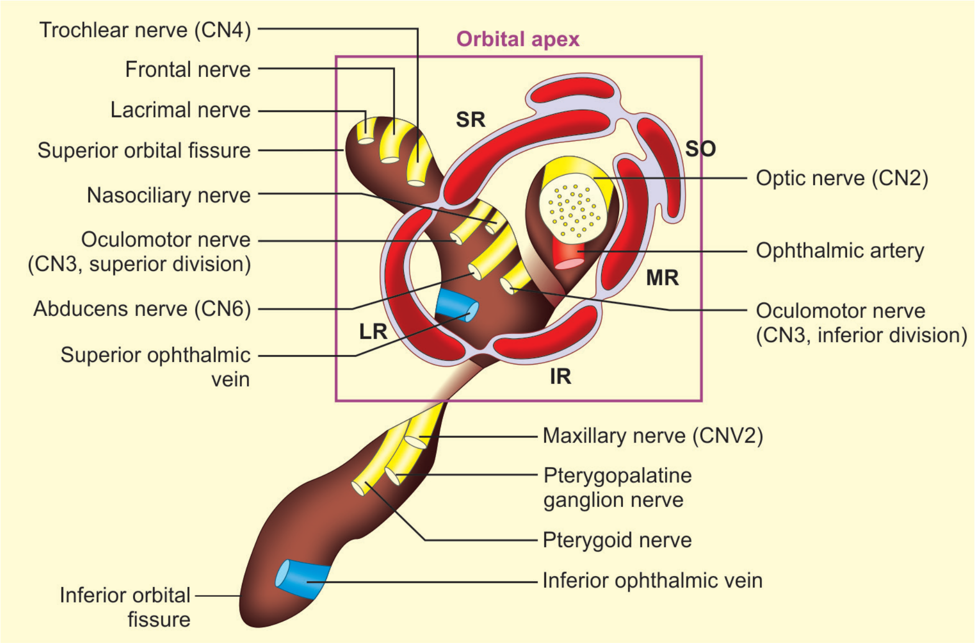

Trochlear nerve (CN4)
Orbital apex
Frontal nerve
Lacrimal nerve
SR
Superior orbital fissure
SO
Optic nerve (CN2)
Nasociliary nerve
Oculomotor nerve (CN3, superior division)
Ophthalmic artery
MR
Abducens nerve (CN6)
Oculomotor nerve (CN3, inferior division)
LR
Superior ophthalmic vein
IR
Maxillary nerve (CNV2)
Pterygopalatine ganglion nerve
Pterygoid nerve
Inferior ophthalmic vein
Inferior orbital fissure

Fig. 8.5.15: Orbital apex

Clinical Implications

- The orbital apex consists of the superior orbital fissure, the inferior orbital fissure, and the optic canal
    - The superior orbital fissure consists of the lacrimal nerve, frontal nerve, and trochlear nerve
    - The optic canal transmits the optic canal and central retinal vessels

  - The inferior orbital fissure transmits the abducens nerve, superior and inferior branches of the oculomotor nerve, and the nasociliary nerve
- As a result, a lesion of the orbital apex is characterized by total ophthalmoplegia, complete ptosis, loss of pupillary reflexes, loss of corneal sensations, and loss of visual acuity
- Common causes of orbital apex syndrome are tumors, infections (mucormycosis), and inflammation (Tolosa Hunt syndrome, orbital pseudotumor)

5. Describe four causes for disc edema. Explain the mechanism of disc edema in hypertensive retinopathy. (4+1 marks)

Causes for Disc Edema

Disc edema is swelling of the axons of the optic nerve head due to many causes

1. **Inflammation**
   - Includes infective and noninfective causes
   - Infective causes: Spread from local sites of infection, para-infectious optic neuropathy, cat-scratch disease, syphilis, Lyme disease, etc.
   - Noninfectious causes: Demyelination like multiple sclerosis, neuromyelitis optica, Chronic relapsing inflammatory optic neuropathy, and autoimmune optic neuritis. It may also be seen in association with posterior scleritis and uveitis
2. **Intracranial pressure**
   - Optic disc edema due to raised intracranial tension is called papilledema
   - It is non-inflammatory disc edema due to axonal stasis
3. **Ischemia**
   - Disc edema is seen in anterior ischemic optic neuropathy due to sudden interruption of blood supply to the optic nerve head by short posterior ciliary arteries
4. **Systemic hypertension**
   - Disc edema is seen in grade IV hypertensive retinopathy and malignant hypertension

Mechanism of Disc Edema in Hypertensive Retinopathy

- Disc edema is seen in grade IV hypertensive retinopathy and malignant hypertension
- This is due to fibrinoid necrosis of the vessels supplying the optic disc causing ischemia and thus giving rise to disc edema

6. Describe the basis, characteristics, and uses of visually evoked potential (VEP). (1+2+2 marks)

VEP

An objective electrophysiological test that tests the integrity of the afferent visual pathway

Basis

- It is a noninvasive test, where surface electrodes are placed on the scalp in the occipital area and visual stimuli are shown to the individual
- The recordings are amplified and demonstrated on the computer screen
- The recordings are unaffected by medial opacities, and thus can be used to prognosticate visual outcomes or understand optic nerve status
- It is nonspecific and cannot localize the lesion

Characteristics

- The normal VEP has an initial negative wave at 75 ms called the N75, an initial positive wave at 100 ms called the p100 and a second negative wave at 135 ms (Fig. 8.5.16)
- The amplitude of the p100 is considered normal if the value is between 10–25 microvolts
- The two methods of performing a VEP are flash VEP and a pattern VEP
- Flash VEP is recorded by using flashes of light released from a shutter and recording the potentials. This gives a rough estimate of the integrity of the optic nerve
- Pattern VEP presents patterned stimuli of decreasing sizes and is useful in the assessment of vision in preverbals

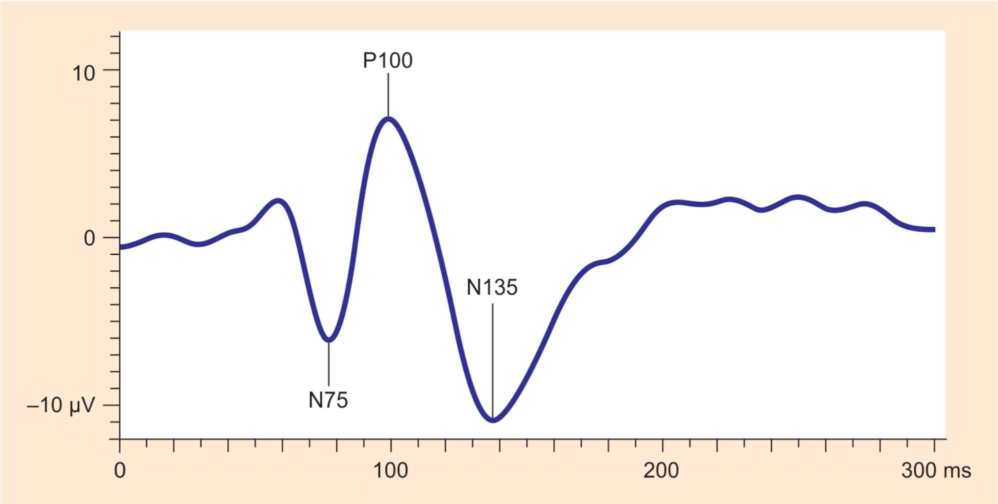


Fig. 8.5.16: Normal VEP

Uses

1. VEP is a test that can objectively distinguish between organic and inorganic causes of loss of vision
2. In optic neuritis, there is a decrease in amplitude and an increase in latency in the early disease when there is active inflammation. After the attack subsides, the latency continues to be increased whereas the amplitude comes back to normal
3. Visual evaluation in infants, comatose patients, inarticulate adults
4. Confirmation of integrity of visual pathway in suspected nonorganic blindness/ malingering

7. Enumerate four congenital disc anomalies and explain the clinical features of morning glory disc syndrome. (2+3 marks)

Congenital Disc Anomalies

1. Optic disc coloboma
2. Hypoplasia of the disc
3. Morning glory syndrome
4. Optic disc drusen

Morning Glory Syndrome

- It is an excavated disc anomaly characterized by a large disc with a central tuft of glial tissue. Sometimes the excavation may be large enough to include the macula (macular capture)
- Spoke-like vessels appear radiating outside like a morning glory flower
- Patients present early in childhood with unilateral poor visual acuity, or strabismus
- A small proportion of patients develop cataracts or nystagmus
- It may be associated with mental retardation
- Associated with transsphenoidal encephalocele, hypertelorism, depressed nasal bridge

8. Compare and contrast true disc edema and pseudo-disc edema. (5 marks)

- Pseudo-disc edema is described in congenital anomalous discs that may have one or more features of disc edema like elevation, blurred disc margins, or filled cup
- The following features help in differentiating the two

Feature	True disc edema	Pseudo-disc edema
Disc color	Hyperemic	Pale yellow
Nerve fiber layer	Opacified due to edema	Transparent
Large vessels	Normal, but maybe tortuous	Anomalous, trifurcation or spoke like
Small vessels	Congested	Normal
Spontaneous venous pulsation	Absent	Present in 80% cases
Nerve fiber hemorrhages	Present	Absent
Hyaline bodies	Absent	May be present
Fluorescein angiogram	Leakage at disc	No leakage

9. Define benign intracranial hypertension. Explain the clinical features. (2+3 marks)

Benign Intracranial Tension/Pseudotumor Cerebrii

Definition

High intracranial pressure in absence of space-occupying lesions, arteriovenous malformations, and obstruction of CSF outflow.

Clinical Features

Symptoms

- Usually present in fertile, fat females in their third or fourth decade with a sudden throbbing headache and projectile vomiting
- They may also present with transient episodes of obscurations of vision lasting for around 30 seconds followed by complete recovery
- These episodes are precipitated by a sudden change in body posture
- They may also present with back pain and pulsatile tinnitus

Signs

- Visual acuity and anterior segment are normal
- Fundus examination reveals features of papilledema
- The disc is elevated to around 2–6 D and shows all the features of papilledema

- Vascular signs
  - Venous congestion of arcuate and peripapillary vessels
  - Papillary and retinal peripapillary hemorrhages
  - Nerve fiber layer infarcts (cotton-wool spots)
  - Hyperemia of the optic nerve head
- Mechanical signs
  - The blurring of the optic disc margins (superonasal and inferonasal are the first to be involved)
  - Filled optic disc cup
  - Elevation of the surface of the disc
  - Edema of the peripapillary nerve fiber layer
  - Paton's lines: Circumpapillary gray folds in the nerve fiber layer
- There are no other signs of focal neurological deficit
- An abducens palsy may, however, be present
- Visual field analysis shows an enlarged blind spot corresponding to the nerve fiber edema
- These episodes may be self-limited and recurring
- If care is not taken to control the intracranial pressure, permanent blindness may occur due to optic atrophy

10. Differentiate between typical optic neuritis and atypical optic neuritis. (5 marks)

Differentiating Features

Typical optic neuritis	Atypical optic neuritis
• Usually, unilateral associated with painful eye movements • Retrobulbar neuritis or minimal optic disc swelling • Affect women in the 2–3rd decade • Usually associated with multiple sclerosis • Rapid onset of visual loss followed by the beginning of recovery by third or fourth week and complete visual recovery after 6 weeks	• Can be bilateral, absence of pain • More pronounced disc edema may be associated with macular star • No gender bias affects any age group • No improvement of vision beyond 2 weeks • Needs to be investigated for other causes like NMo-IgG (neuromyelitis optica), sarcoid, autoimmune optic neuropathy, and chronic relapsing inflammatory optic neuropathy. Infections also need to be considered

11. Describe the clinical features of anterior ischemic optic neuropathy. (5 marks)

Anterior Ischemic Optic Neuropathy (AION)

- This is optic neuropathy occurring due to ischemia of the optic nerve head due to interruption of vascular supply—short posterior ciliary vessels
- It is the most common form of optic neuropathy occurring in patients above the age of 50 years

Symptoms

- The patients usually present with sudden painless monocular loss of vision developing hours to days
- If it is associated with giant cell arteritis, they may present with jaw claudication and unilateral headache

Signs

- Visual acuity is diminished, and a visual field defect is almost always present and is altitudinal

- It can be either superior or inferior
- Signs include RAPD and optic disc edema
- The disc is hyperemic and shows areas of pallor
- Segmental disc edema is pathognomonic of AION
- There can be associated flame-shaped hemorrhages and arterial narrowing in the peripapillary area

Specific features of arteritic variant	Specific features of non-arteritic variant
• More in elderly women (7–8th decade) • Associated with giant cell arteritis • May have premonitory signs like one-sided headache, jaw claudication, and bright light-induced amaurosis • Visual loss is more severe • The disc shows pallid edema • It is very much responsive to systemic corticosteroids and	• No gender predilection, 5th or 6th decade • Associated with a crowded optic disc • Sudden painless diminution of vision • Increased rate of fellow eye involvement

12. A 30-year-old man is brought to the emergency room in a semiconscious state and is moaning in pain. He has a strong formalin odor from his mouth. The bystanders gave a history of ingestion of country liquor.

A. What is the probable diagnosis? (1 mark)
B. Explain the ocular manifestations of this condition. (2 marks)
C. Explain the treatment of this condition. (2 marks)

A. Most Probable Diagnosis

Methanol poisoning

B. Ocular Manifestations

- Methanol poisoning is usually caused by the ingestion of methylated spirit, inhalation of fumes, and suicidal attempts; and is an extremely dangerous condition and is associated with significant mortality
- The methanol is converted into formaldehyde and formalin. These chemicals are toxic to the ganglion cells, and papillomacular bundles are most vulnerable
- Visual acuity usually cannot be assessed due to the semiconscious state
- Pupillary reactions are sluggish
- There is diffuse neuronal damage and in the early stages, there is mild disc edema
- Within 2 days optic atrophy ensues and visual acuity is severely and irreversibly reduced

C. Management

- It is mainly aimed at the removal of methanol and correction of acid-base imbalance (metabolic acidosis)
- Fomepizole is an inhibitor of alcohol dehydrogenase and can be given to block the conversion of methanol to formalin
- Another alternative is ethanol. However, the dosing of the former is easier and free of side effects
- Gastric lavage is done to remove the ingested methanol
- Intravenous bicarbonate to normalize the acidity
- Peritoneal dialysis or hemodialysis: As methanol and formate are both non-protein bound, they are easily dialysable

13. Describe the etiology and clinical features of chiasmal syndrome. (2+3 marks)

Chiasmal Syndrome

A constellation of signs and symptoms that occur due to compression of the optic chiasm.

Etiology

A. *Compression from superior aspect*
   1. Dilatation of the third ventricle
   2. Gliomas
   3. Ectopic pinealomas
   4. Suprasellar meningiomas

B. *Compression from inferiorly*
   1. Tumors of the pituitary

C. *Compression from laterally*
   1. Craniopharyngioma
   2. Rathke's pouch remnants
   3. Germ cell tumor
   4. Cavernous sinus thrombosis

Clinical Features

1. **Ocular features**
   - Decreased visual acuity
   - Dyschromatopsia
   - Pupillary field defects include RAPD and APD
   - Visual field defects include bitemporal hemianopia, junctional scotoma, binasal hemianopia, and bitemporal hemianopic scotoma depending on the site of compression
   - Optic disc reveals bow-tie atrophy due to compression of nasal retinal and nasal macular fibers
   - See-saw nystagmus
2. **Systemic features**
   - Headache
   - Hypersecretion of pituitary hormones and their resultant effects

14. Describe the arrangement of nerves in the optic chiasma. Explain junctional scotoma and the anatomical basis for it. (2+3 marks)

Arrangement of Nerves at the Optic Chiasma (Fig. 8.5.17)

- Inferior and nasal fibers are the anterior-most
- The superior nasal fibers are the posterior-most and travel high in the chiasm
- The temporal fibers remain uncrossed and traverse through the lateral part
- Macular fibers usually occupy the central part of the optic nerve, but in the chiasma, nasal fibers cross to the opposite

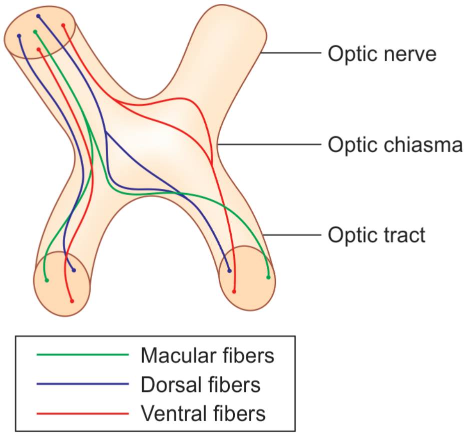


Fig. 8.5.17: Arrangement of fibers in the optic chiasm

Junctional Scotoma

- It is characterized by ipsilateral central scotoma and contralateral scotoma in the superotemporal quadrant (Fig. 8.5.18)
- The superotemporal scotoma does not have a proper shape (as in a typical bitemporal hemianopia lesion)
- This is called a junctional scotoma as the site of the lesion is at the junction of the optic nerve and the chiasm

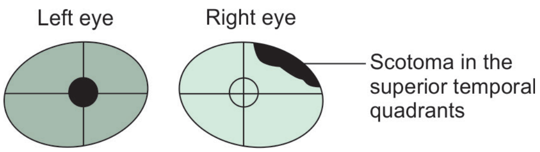

Left eye
Right eye
Scotoma in the superior temporal quadrants

Fig. 8.5.18: Junctional scotoma

Anatomical Basis for Junctional Scotoma

- Junctional scotomas are seen in patients with olfactory meningiomas and meningiomas of the sphenoidal margin
- These tumors compress the anterior-most part of the chiasm, where the inferonasal fibers of the contralateral optic nerve enter the ipsilateral optic nerve for a short distance (von Willebrand's knee) and then proceed towards the optic chiasm. (Fig. 8.5.19)

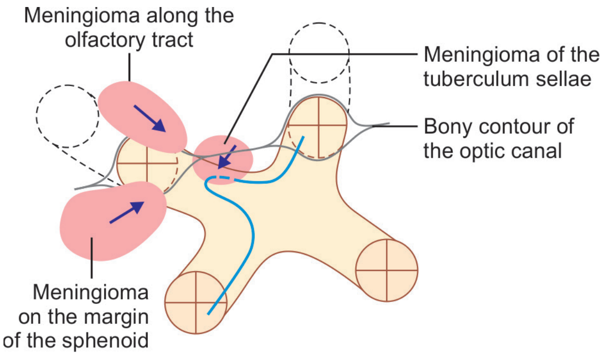

Meningioma along the olfactory tract
Meningioma of the tuberculum sellae
Bony contour of the optic canal
Meningioma on the margin of the sphenoid

Fig. 8.5.19: Causes for junctional scotoma

- Other features of sphenoid wing meningiomas include fullness in the temporal fossa, eccentric proptosis, and ophthalmoplegia depending on the extent and position of the tumor

15. Describe the characteristics of visual field defects that can be seen with chiasmal compression. (5 marks)

Field Defects Associated with Chiasmal Compression

1. **Bitemporal superior quadrantanopia (Fig. 8.5.20)**
   - Mass arising from the pituitary compresses the chiasma from its anterior and inferior part (Fig. 8.5.21)
   - The result is bitemporal hemianopia that starts initially as a bitemporal superior quadrantanopia and then progresses inferiorly to involve the lower field
   - The filed defect is denser superiorly
2. **Bilateral inferior quadrantanopia (Fig. 8.5.22)**

- Tumors like craniopharyngioma arise from the pituitary stalk (Rathke's pouch derivatives) (Fig. 8.5.23)
- These lesions cause compression of the chiasm from the superior and posterior aspects
- They also cause bitemporal hemianopia which begins as bitemporal inferior quadrantanopia and then the field defect progresses superiorly

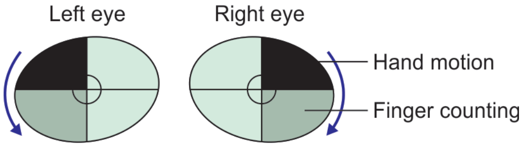

Left eye
Right eye
Hand motion
Finger counting

Fig. 8.5.20: Superior quadrantanopia

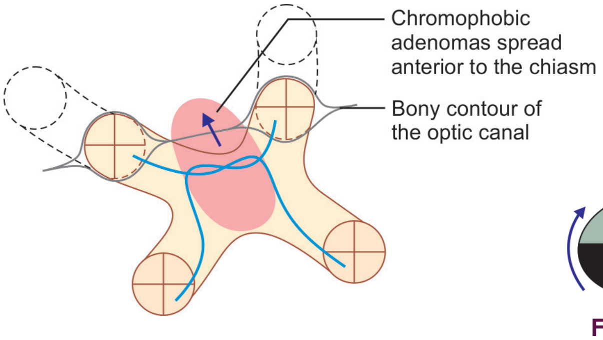

Chromophobic adenomas spread anterior to the chiasm
Bony contour of the optic canal

Fig. 8.5.21: Pituitary mass compressing fibers in the optic chiasm anteriorly

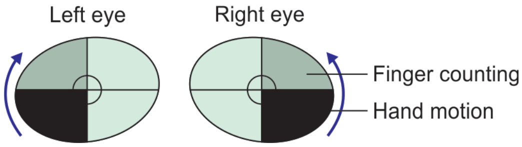

Left eye
Right eye
Finger counting
Hand motion

Fig. 8.5.22: Bilateral inferior quadrantanopia

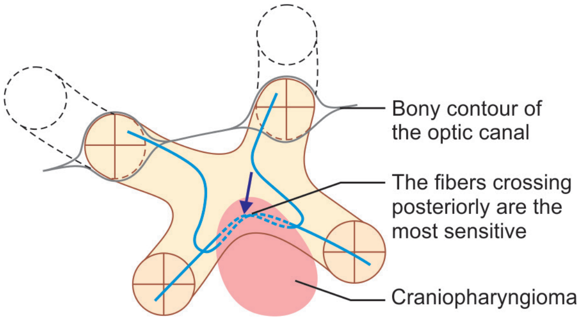

Bony contour of the optic canal
The fibers crossing posteriorly are the most sensitive
Craniopharyngioma

Fig. 8.5.23: Tumors compressing the chiasm from posteriorly

3. **Junctional scotoma**
   - This is called a junctional scotoma as the site of the lesion is at the junction of the optic nerve and the chiasm
   - It is characterized by ipsilateral central scotoma and contralateral scotoma in the superotemporal quadrant. The superotemporal scotoma does not have a proper shape (as in a typical bitemporal hemianopia lesion) (Fig. 8.5.24)
   - Tumor compressing the chiasm in the anterior aspect
   - These tumors compress the anterior-most part of the chiasm, where the inferonasal fibers of the contralateral optic nerve enter the ipsilateral optic nerve for a short distance (von Willebrand's knee) and then proceed towards the optic chiasm (Fig. 8.5.25)

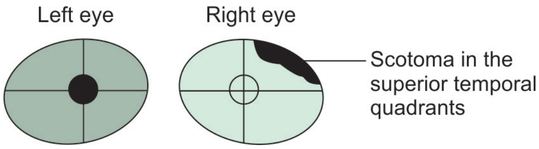

Left eye
Right eye
Scotoma in the superior temporal quadrants

Fig. 8.5.24: Junctional scotoma

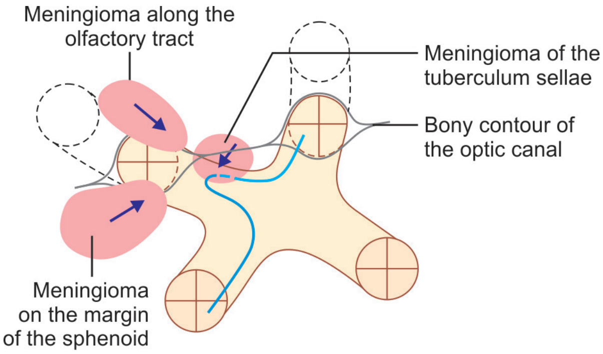

Meningioma along the olfactory tract
Meningioma of the tuberculum sellae
Bony contour of the optic canal
Meningioma on the margin of the sphenoid

Fig. 8.5.25: Tumor compressing the chiasm in the anterior aspect

4. **Binasal hemianopia**
   - This is called binasal hemianopia, which is denser on the side of the compression (Fig. 8.5.26)

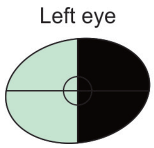

Left eye
Right eye

Fig. 8.5.26: Binasal hemianopia

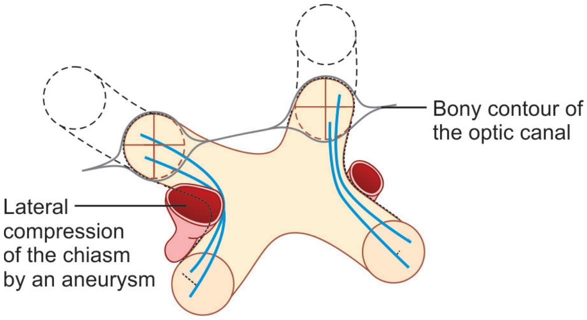

Bony contour of the optic canal
Lateral compression of the chiasm by an aneurysm

Fig. 8.5.27: Laterally compressing tumors

- Laterally compressing tumors: Diseases of the internal carotids like atherosclerosis or aneurysms cause lateral compression of the chiasm causing compression of the uncrossed temporal fibers (Fig. 8.5.27)

16. Explain the pupillary light reflex pathway with a neat and labeled diagram. (5 marks)

Pupillary Light Reflex (Fig. 8.5.28)

- When light is shone into one eye, contraction of pupils occurs in both eyes: This is called light reflex
- It is called direct when the ipsilateral pupil reacts and consensual when the contralateral pupil reacts

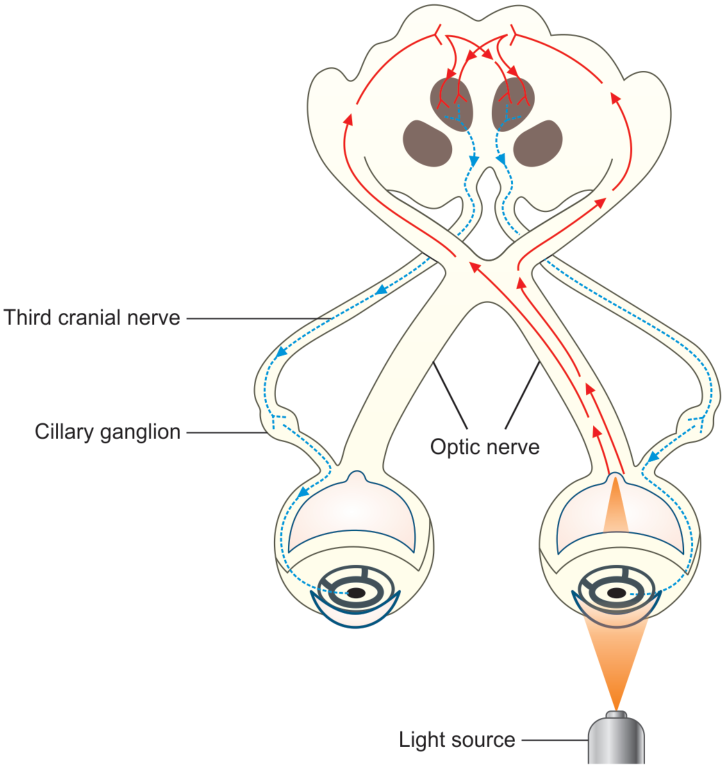
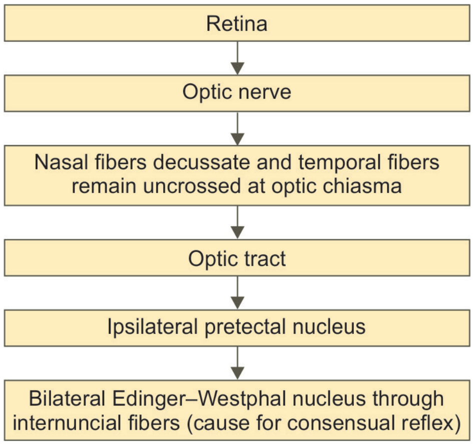
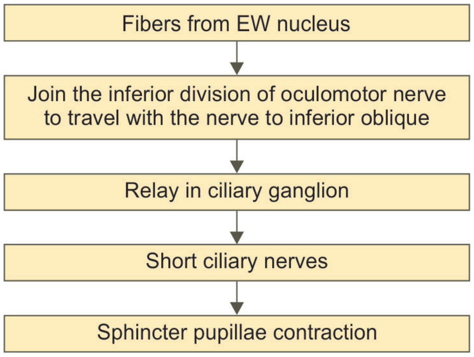

Third cranial nerve
Cillary ganglion
Optic nerve
Light source

Fig. 8.5.28: Pathway of light reflex

Afferent


Retina
Optic nerve
Nasal fibers decussate and temporal fibers remain uncrossed at optic chiasma
Optic tract
Ipsilateral pretectal nucleus
Bilateral Edinger–Westphal nucleus through internuncial fibers (cause for consensual reflex)

Efferent


Fibers from EW nucleus
Join the inferior division of oculomotor nerve to travel with the nerve to inferior oblique
Relay in ciliary ganglion
Short ciliary nerves
Sphincter pupillae contraction

17. What is a near reflex? What are its components and explain the pathway? (1+1+3 marks)

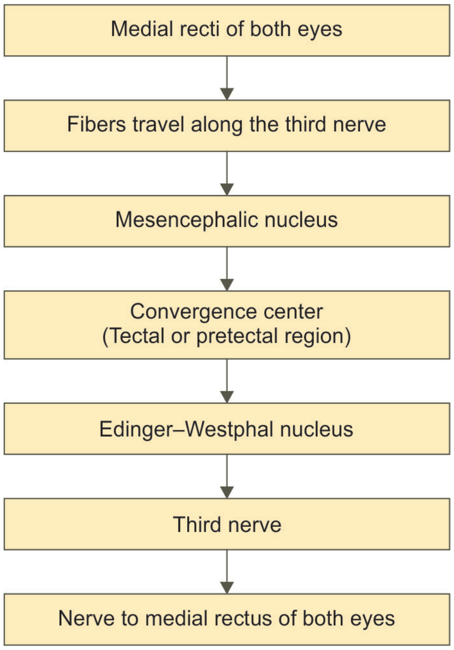
Near Reflex

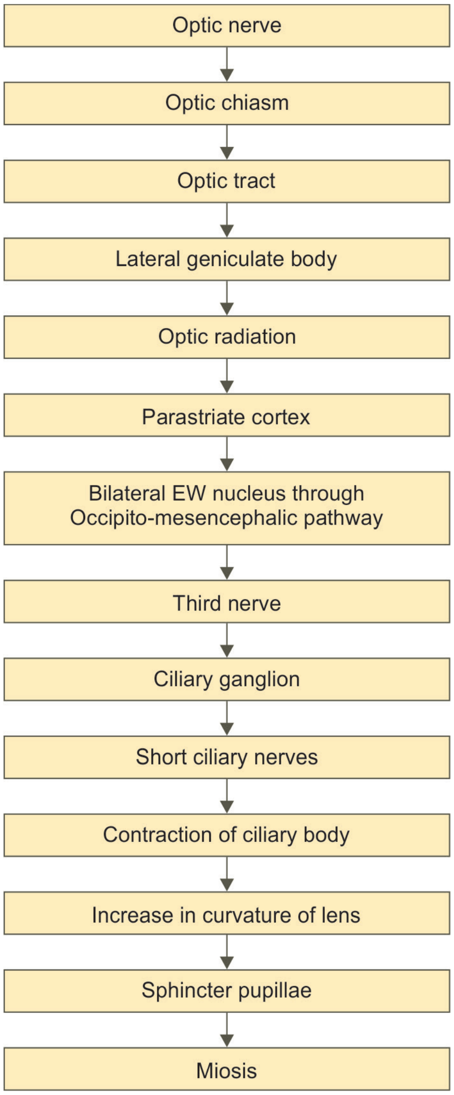
An involuntary reflex that occurs to bring near objects into focus.

Components

1. Convergence of the eyeballs
2. Increase in the anterior curvature of the lens
3. Contraction of bilateral pupils

Pathway for Convergence


Medial recti of both eyes
Fibers travel along the third nerve
Mesencephalic nucleus
Convergence center (Tectal or pretectal region)
Edinger–Westphal nucleus
Third nerve
Nerve to medial rectus of both eyes


Optic nerve
Optic chiasm
Optic tract
Lateral geniculate body
Optic radiation
Parastriate cortex
Bilateral EW nucleus through Occipito-mesencephalic pathway
Third nerve
Ciliary ganglion
Short ciliary nerves
Contraction of ciliary body
Increase in curvature of lens
Sphincter pupillae
Miosis

18. Explain the terms afferent pupillary defect and efferent pupillary defect and differentiate between the two. (1+1+3 marks)

Afferent Pupillary Defect

- The afferent pupillary defect occurs due to a lesion in the afferent pupillary pathway
- It is characterized by a sluggish direct response of the ipsilateral eye and sluggish consensual reflex of the contralateral eye
- It is defined as an apparent dilatation of the pupil of the eye with a defective afferent pathway when light is swung from the normal eye to the abnormal eye

Efferent Pupillary Defect

- It is due to lesions involving the efferent pathway leading to the abolition of light reflex and near reflex on the ipsilateral side
- Both the reflexes are present on the contralateral side. As a result, there is variation in the size of the pupil (anisocoria)

Differences between APD and EPD

<table>
<tr><th>Feature</th><th>Afferent pupillary defect</th><th>Efferent pupillary defect</th></tr>
<tr><td>Location of lesion</td><td>From retina to the optic tract</td><td>From pretectal nucleus to sphincter pupillae</td></tr>
<tr><td>Size of pupil</td><td>Same in both eyes</td><td>Anisocoria</td></tr>
<tr><td>Direct and consensual response</td><td>Direct is sluggish/absent in the ipsilateral eye
Consensual is sluggish/absent in the contralateral eye</td><td>Direct and consensual are sluggish/absent on the affected side</td></tr>
<tr><td>Example Lesion of the right eye</td><td>Right APD
<table>
<tr><td>Light on the right eye</td><td>Right eye pupil 4 mm</td><td>Left eye pupil 4 mm</td></tr>
<tr><td>Light on the left eye</td><td>Right eye pupil 2 mm</td><td>Left eye pupil 2 mm</td></tr>
</table></td><td>Right EPD
<table>
<tr><td>Light on the right eye</td><td>Right eye pupil 6 mm</td><td>Left eye pupil 3 mm</td></tr>
<tr><td>Light on the left eye</td><td>Right eye pupil 6 mm</td><td>Left eye pupil 3 mm</td></tr>
</table></td></tr>
</table>

19. Enumerate the causes for a relative afferent pupillary defect. (5 marks)

1. **Retina**
   i. Central retinal artery obstruction
   ii. Ischemic central retinal vein obstruction
   iii. Ocular ischemic syndrome
   iv. Huge retinal detachment
   v. Severe macular degeneration
   vi. CMV retinitis
2. **Optic nerve**
   i. Optic neuritis
   ii. Optic atrophy
   iii. Compressive optic neuropathy
   iv. Ischemic optic neuropathy
   v. Traumatic optic neuropathy
   vi. Glaucomatous optic atrophy
3. **Optic chiasm and optic tract**
   i. Compressive lesions
   ii. Vascular lesions
   iii. Traumatic lesions
   iv. Demyelination
4. **Pretectal**
   i. Ischemic lesions
   ii. Trauma
   iii. Neoplasia

20. Describe the causes of Efferent Pupillary Defects (EPD). (5 marks)

Causes of EPD

- Lesions of the superior colliculus, third nerve palsy, damage to the ciliary ganglion, and short ciliary nerves, damage to sphincter pupillae, and the use of mydriatics

- Physiological anisocoria is a difference of 1 mm between the two pupils which is equal in bright and dim illumination

Causes for anisocoria worsening in dark	Causes for anisocoria worsening in bright light
• Physiological • Horner's syndrome	• Adie's pupil • Third nerve palsy • Drug-induced mydriasis

Horner Syndrome

- It is oculosympathetic paralysis characterized by unilateral miosis, dilatation lag of pupil in dark, mild ptosis (due to a weakness of Muller's muscle), enophthalmos, anhidrosis
- In congenital Horner's syndrome, the affected iris is heterochromic

Adie's Pupil

- It is due to inflammation in the ciliary ganglion leading to neuronal loss
- It is a syndromic association with absent deep tendon reflexes and orthostatic hypotension, usually seen in women, also called "Holmes–Adie syndrome"
- The involved pupil is larger than the normal one and light and near reflexes are sluggish
- On slit-lamp examination, one can see a segmental contraction of the pupillary sphincter and thinning of the iris at areas where there is no contraction
- They show supersensitivity to 0.1% pilocarpine compared to the normal eye

Third Nerve Palsy

- The involved pupil is larger than the fellow pupil
- It constricts when pilocarpine is instilled

Drug-induced Mydriasis

- In this, the affected pupil is larger than the other and is insensitive to the application of pilocarpine
- It returns to normal after stipulated amount of time depending on the mydriatic used

21. Enumerate the muscles supplied by the oculomotor nerve. Explain the features of aberrant regeneration of the third nerve. (3+2 marks)

Muscles Supplied by the Superior Branch of the Oculomotor Nerve

1. Levator palpebrae superioris
2. Superior rectus

Muscles Supplied by the Inferior Branch of the Oculomotor Nerve

1. Inferior rectus
2. Medial rectus
3. Inferior oblique
4. Sphincter pupillae

Aberrant Regeneration of the Oculomotor Nerve

- Also called oculomotor synkinesis

- Usually seen during the healing of traumatic or inflammation of the third nerve
- It is characterized by haphazard redirection of repairing neurons because of which there are undesired effects
- Examples are:
  1. Pseudo-von Graeffe phenomenon: Retraction of the upper lid on attempted downgaze
  2. Adduction of the eye on attempted upward or downward gaze
  3. Constriction of the pupil on attempted adduction (pseudo-Argyll Robertson pupil)

22. Enumerate the syndromes associated with fascicular lesions of the third nerve. (5 marks)

Syndromes Associated with Fascicular Lesions

Syndrome	Site of lesion	Features
Benedict syndrome (dorsal fascicular)	Lesion while passing through the red nucleus	Ipsilateral 3rd nerve palsy + contralateral tremor
Weber syndrome (ventral fascicular)	Lesion while passing through cerebral peduncle)	Ipsilateral 3rd nerve palsy + contralateral hemiplegia
Nothnagel syndrome	Involves fasciculus and superior cerebellar peduncle	Ipsilateral 3rd nerve palsy + cerebellar symptoms
Claude syndrome	Benedict + Nothnagel syndromes	

23. A woman in her 6th decade presents with sudden diplopia and severe headache. On examination blood pressure is 180/110 mm Hg, and there is moderate ptosis of her right eye with the eye deviated out and down.

A. State the most probable diagnosis? (1 mark)

B. Explain the role of the pupil in distinguishing the cause for the same? (1 mark)

C. How do you differentiate superior orbital fissure syndrome, orbital apex syndrome, and cavernous sinus syndrome? (3 marks)

A. Most Probable Diagnosis

- A right-sided isolated third nerve palsy
- Whether the palsy is involving the pupil or not is not clear from the case scenario

B. Role of Pupil in Third Nerve Palsy

- The most common cause for pupil-sparing third nerve palsy is diabetes mellitus
- The pupillomotor fibers are more superficial and are supplied from pial vessels and hence spared in an ischemic event that usually involves the vasa nervorum
- Involvement of pupil warrants immediate neuroimaging to rule out compressive lesions like posterior communicating artery aneurysm
- As pupillomotor fibers are more superficial, the pupil is always involved in compressive or 'surgical' causes

C. Differences between Superior Orbital Fissure Syndrome, Orbital Apex Syndrome, and Cavernous Sinus Syndrome

The differentiation among these three is required as all three-cause total ophthalmoplegia, with complete ptosis, and loss of pupillary reflex

Superior orbital apex syndrome	Orbital apex syndrome	Cavernous sinus syndrome
• Total ophthalmoplegia • Loss of corneal reflex • No visual loss • No RAPD	• Total ophthalmoplegia • Loss of corneal reflex • Visual loss • RAPD	• Total ophthalmoplegia • Loss of corneal reflex • Visual loss • RAPD • Loss of sensations over the maxillary region due to the affection of the V2 branch of the trigeminal nerve

24. Describe the supranuclear control of eye movements. (5 marks)

Supranuclear Control of Eye Movements

- Eye movements are of two types: Saccades and pursuits
- Saccades are fast fixating movements. Pursuits are slow, following movements of the eye

Horizontal Gaze Control

- Horizontal saccades are initiated in the contralateral frontal cortex and parietal eye field. Connections from here pass through the superior colliculus to enter the horizontal gaze center
- The pathway for horizontal gaze arises from the ipsilateral 6th nerve and neurons reach the parapontine reticular formation (PPRF) on the same side, fibers from which supply the ipsilateral lateral rectus (Fig. 8.5.29)
- Whereas internuclear neurons cross the midline at the level of pons and relay in the third nerve nucleus and then supply the contralateral medial rectus
- Thus, for the right gaze, the 6th nerve and PPRF on the same side are stimulated

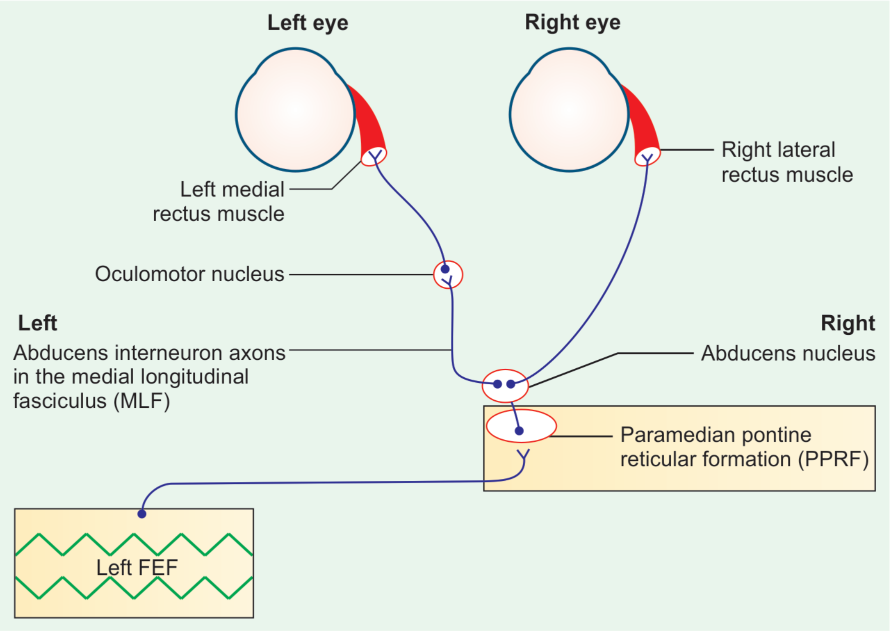

Left eye
Right eye
Right lateral rectus muscle
Left medial rectus muscle
Oculomotor nucleus
Left
Abducens interneuron axons in the medial longitudinal fasciculus (MLF)
Right
Abducens nucleus
Paramedian pontine reticular formation (PPRF)
Left FEF

Fig. 8.5.29: Horizontal gaze control

Vertical Gaze Control

- Vertical saccades are initiated in the ipsilateral frontal eye field and parietal eye field
- Vertical gaze control is in the rostral interstitial medial longitudinal fasciculus. It is a bilateral structure connected through the posterior commissure
- They connect to the oculomotor and the trochlear nerve nuclei
- Pursuits are controlled by the ipsilateral occipital cortex. These movements help in following moving objects
- The interstitial nucleus of Cajal is involved in vertical gaze holding and pursuit

25. Describe the pathophysiology and clinical features of migraine. (2+3 marks)

Migraine

Pathophysiology (Fig. 8.5.30)

- The etiology of migraine was thought to be vascular in origin
- It has a strong genetic component and thus patients have a family history
- It is thought to arise from the brainstem followed by vasoconstriction or vasodilatation of the intracranial and extracranial arteries. Then there is an activation of the trigeminal pain center
- The large dural vessels are surrounded by the ophthalmic division of the trigeminal nerve and in the posterior fossa, by the dorsal root nerves

Clinical Features

- Migraine is present in around 10% of the population and affects women more
- There is usually a strong family history, and the age of onset is usually in the second-fourth decade
- Usual triggers are sleeplessness, stress, menstruation, alcohol, caffeine (sometimes), chocolate ingestion and varies with individuals
- A typical attack begins with a visual or auditory hallucination which is called the aura. The common visual aura is the fortification spectrum where the patient sees colorful

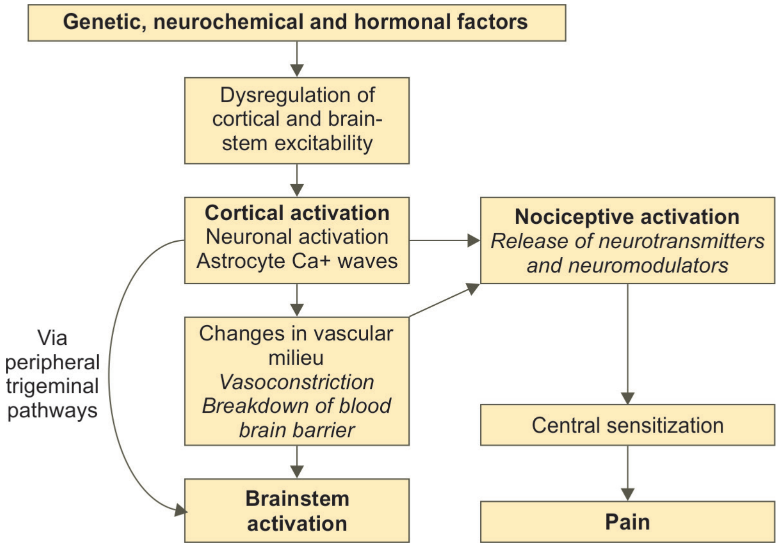

Genetic, neurochemical and hormonal factors
Dysregulation of cortical and brain-stem excitability
Cortical activation
Neuronal activation
Astrocyte Ca+ waves
Nociceptive activation
Release of neurotransmitters and neuromodulators
Via peripheral trigeminal pathways
Changes in vascular milieu
Vasoconstriction
Breakdown of blood brain barrier
Central sensitization
Brainstem activation
Pain

Fig. 8.5.30: Pathophysiology of migraine

rainbow-like images which begin in the periphery like a fort and is made of zigzag lines
- The headache is usually unilateral, in the frontotemporal region, and associated with nausea and vomiting. The individual prefers to be in darkness and avoid the sound
- Sometimes there may be associated third nerve palsy (ophthalmoplegic migraine)

SHORT ANSWERS

1. Explain the diagnostic criteria for idiopathic intracranial hypertension. (3 marks)

Modified Dandy's criteria for the diagnosis of IIH is as follows:
1. Features of raised intracranial pressure like headache, projectile vomiting, and papilledema. CSF opening pressure >200 cm of H_2O
2. Absence of focal neurological deficits except for abducens palsy
3. Normal CSF analysis and normal neuroimaging excepting a decrease in the size of the ventricles

2. Enumerate three tests used for color vision testing. (3 marks)

1. Ishihara's pseudoisochromatic charts (most used)
2. Holmgren's wools test
3. Hardy Rand Rittler charts (used to detect congenital color blindness)
4. Farnsworth Munsell 100 hue test (most sensitive)

3. Explain the clinical features of tilted disc anomaly. (3 marks)

- The optic nerve enters the eyeball in an oblique fashion leading to posteriorly placed inferonasal margin and anteriorly placed superotemporal margin
- The disc appears oblong, and vessels appear to arise from the nasal side (situs inversus of the blood vessels)
- Along with the above two features, there is inferonasal chorioretinal thinning and myopic astigmatism
- Field analysis shows superotemporal quadrantanopia and may be confused with chiasmal lesions, but these defects do not respect the vertical midline as neurological lesions do

4. Explain the disc features of early papilledema. (3 marks)

Early papilledema is characterized by:
1. Blurring of the superonasal and the inferonasal margins
2. There is a mild elevation of the nerve fiber layer
3. Loss of venous pulsations
4. Hyperemia of the disc surface

5. Describe the fundus characteristics of compressive optic neuropathy. (3 marks)

Fundus Examination Reveals

- There is disc edema in cases of acute compression (orbital compartment syndrome), early stages, and anterior orbital lesions due to venous stasis
- Other features of optic neuropathy are present (decreased visual acuity, RAPD, dyschromatopsia)
- Disc edema is accompanied by optico-ciliary shunts which bypass retinal circulation into the ciliary circulation
- However, the phase of disc edema is short-lived and then optic atrophy ensues

6. Explain the clinical features of traumatic optic neuropathy. (3 marks)

Clinical Features of Traumatic Optic Neuropathy

- Visual acuity may vary from 6/12 to negative perception of light
- The most objective and reliable sign is an afferent pupillary defect
- Fundus signs vary depending on the site of trauma
  - *Optic nerve head avulsion*
    - Characterized by partial/complete avulsion of the optic nerve from lamina cribrosa
    - Fundus shows a ring of hemorrhages around the disc area
  - *Anterior optic neuropathy*
    - Occurs within 10 mm of entry of the central retinal artery
    - It may present as CRAO, CRVO, or AION
  - Posterior optic neuropathy
    - Injury occurs behind the entry of the central retinal artery
    - The fundus is normal in acute stages but optic atrophy sets in later
    - This is the most common presentation
- May be associated with other ocular injuries

7. Define Foster Kennedy syndrome? Explain the anatomical basis for the same. (1+2 marks)

Foster Kennedy Syndrome

Definition

Characterized by ipsilateral optic atrophy and contralateral papilledema.

Anatomical Basis

Arises due to tumor of the anterior cranial fossa (olfactory groove meningioma, frontal lobe tumor) compressing the ipsilateral optic nerve causing primary optic atrophy and large enough to cause raised intracranial tension causing contralateral papilledema is caused by raised intracranial pressure due to the tumor.

8. Explain the terms (1 mark each)

A. Latency in VEP

B. Amplitude in VEP

C. Uhthoff phenomenon

A. Latency

- It is the period after which the first positive wave is seen in a pattern reversal VEP
- It is normally 100 ms
- It is increased in demyelinating disorders

B. Amplitude in VEP

- It is the height of the first positive wave in a pattern reversal VEP
- Its value is between 10–25 mV

C. Uhthoff Phenomenon

- It is visual obscuration during a hot shower or exercise causing increased body temperature
- It is characteristic of a demyelinating disorder

9. Explain the anatomical basis for macular sparing in occipital lesions. (3 marks)

- In occipital lesions due to posterior cerebral artery infarct, there is a contralateral congruous homonymous hemianopia with the sparing of the macula
- The reasons for this are:
  1. The macula is represented by the tip of the visual cortex has a dual blood supply from twigs of the posterior cerebral artery and the middle cerebral artery
  2. Macula has bilateral representation

10. Explain, how do you differentiate lesions of the optic tract from those of optic radiations based on clinical features? (3 marks)

- Both optic tract lesions and optic radiation lesions cause contralateral homonymous hemianopia
- The differences are:
  1. Optic tract lesions are associated with pupillary abnormalities like Wernicke's hemianopic pupil and temporal pallor on the side of lesions and bow-tie atrophy on the other side
  2. Congruity is higher in the field defects due to lesions of optic radiations
  3. Optic tract lesions may be associated with ipsilateral third nerve palsy and contralateral hemiplegia
  4. Lesions of the optic radiations may be associated with auditory hallucinations and spatial disorientation

11. Define light-near dissociation? Give two examples for the same. (1+2 marks)

Light Near Dissociation

Definition

The absence of light reflex of the pupil with an intact near reflex.

Examples

1. Argyll Robertson pupil
2. Dorsal midbrain syndrome

12. Describe the clinical features of Argyll Robertson's pupil. (3 marks)

- In the late 1800s, Argyll Robertson's pupil was described in patients with Tabes Dorsalis, a manifestation of neurosyphilis, and hence was also termed as 'prostitutes' pupil'
- It is also seen in demyelination due to the affection of the pretectal nucleus

Symptoms

There are no symptoms

Signs

- It is characterized by light near dissociation: The absence of light reflex and the presence of near reflex
- The affected pupil is usually smaller than the fellow pupil
- It is usually bilateral, and the pupils are smaller than normal and are unaffected by mydriatics

13. Explain the terms (1 mark each)

A. Hippus
B. Miosis
C. Mydriasis

A. Hippus

It is a spasmodic, rhythmic, and regular constriction and dilatation of the pupil that occurs under physiological circumstances

B. Miosis

it is an abnormal decrease in the size of the pupil (<2 mm)

C. Mydriasis

It is an abnormally increased size of the pupil (>5 mm)

14. Explain the terms (1 mark each)

A. Adie's pupil
B. Anisocoria
C. Hutchison's pupil

A. Adie's Pupil

- It is a type of light-near reflex dissociation. In this, the affected pupil is larger than usual and does not constrict to light. But near reflex is present
- On slit-lamp examination, vermiform movements of the pupil are seen
- After prolonged exposure to light, there is tonic or slow constriction of the pupil followed by a slow relaxation

B. Anisocoria

- It is the difference in the size of both pupils >2 mm

C. Hutchison's Pupil

- In cases of head trauma and following subdural hematoma, there is raised intracranial tension and uncal herniation that leads to the compression of the third nerve over the tentorial edge
- This causes irritative miosis followed by mydriasis and total third nerve palsy

15. Define Wernicke's hemianopic pupil? Explain the cause for the same. (1+2 marks)

Wernicke's Hemianopic Pupil

Definition

- An asymmetric pupillary reaction in a single eye
- It is easily elicited by a narrow-slit beam

Causes

1. It is seen in lesions of the optic tract as the nasal pupillary fibers decussate and the temporal pupillary fibers remain uncrossed
2. In one-sided optic tract lesions, the ipsilateral temporal pupillary fibers and contralateral nasal pupillary fibers are affected (Fig. 8.5.31)

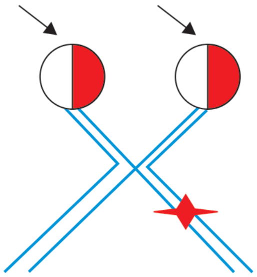


Fig. 8.5.31: Wernicke's hemianopic pupil—site of lesion

16. Demonstrate the knowledge of performing the swinging flashlight test. (3 marks)

- The swinging flashlight test is a simple bedside test to look for pupillary light reflexes
- It is performed usually in mesopic conditions or dim illumination, darkroom is not a prerequisite
- The patient is advised to look at a distant target to avoid eliciting a near reflex and the examiner always stands on either side, again not to elicit a near reflex
- Avoid taking the flashlight too close to the eye as it may elicit a menace reflex
- The flashlight, either incandescent or LED, is shone in the same quadrant in both eyes. For example, if the light is shone from the temporal aspect (more commonly) in the right eye, it should be shone from the temporal aspect in the left eye too
- In this way, all confounders can be easily overcome, and pupillary testing becomes very objective

17. There is anisocoria in an afferent pupillary defect. Is this statement true or false? Justify your statement. (3 marks)

This Statement is False

There is no anisocoria in afferent pathway defect.

Justification

- When light is shone on one eye (say the affected side), there is an equal amount of constriction of both pupils due to the decussation of nerve fibers at the Edinger–Westphal nucleus. When light is shone on the normal side, there is equal and more constriction of both pupils
- In both cases, the pupils remain of equal size and thus there is no anisocoria

18. Hypothesize for each of the following (1 mark each)

A. Lesions beyond the lateral geniculate body do not cause RAPD.
B. A dense cataract or a hyphaema does not cause RAPD.
C. Pupillary light reaction remains the same intensity irrespective of the number of times it is elicited.

A. Lesions beyond the Lateral Geniculate Body do not Cause RAPD

- The reason for this is that the nerve fibers beginning from the lateral geniculate bodies are third-order neurons
- The second-order neurons relay at the LGB
- So, there is no pupillary defect or optic disc pallor in lesions behind the lateral geniculate body

B. A Dense Cataract or a Hyphaema does not Cause RAPD

- Anterior segment lesions like dense cataracts or hyphema do not interfere with the light rays stimulating retinal ganglion cells
- Hence, there is no RAPD due to a dense cataract or hyphema

C. Pupillary Light Reaction Remains the Same Intensity Irrespective of the Number of Times it is Elicited

- The opsin involved in the conductance of impulses for the pupillary pathway is melanopsin, and it cannot be bleached
- As a result, the intensity of the pupillary reaction remains the same irrespective of the number of times it is tested

19. Isolated sixth nerve palsy is a false localizing sign. Is it true? Justify your statement. (3 marks)

Yes, the statement is true

Justification

- Isolated sixth nerve palsy is seen in lesions of the petrous apex. However, more frequently isolated sixth nerve palsy is caused by raised intracranial tension (due to any cause) as the course of the abducens nerve is long and the nerve gets compressed against the petrous apex
- Thus, sixth nerve palsy is not only caused by lesions of the petrous apex. It is a false localizing sign

20. Describe the clinical features of dorsal midbrain syndrome. (3 marks)

Dorsal Midbrain Syndrome

A type of vertical gaze palsy and occurs due to pinealoma, aqueductal stenosis, meningitis, demyelination, trauma, cerebrovascular accidents, posterior fossa aneurysms, and arteriovenous malformations.

Clinical Features

- There is light near dissociation (absence of light reflex and presence of near reflex)
- Eyes are straight in primary position
- Vertical gaze palsy on saccadic movements
- Defective convergence
- Convergence retraction nystagmus (on an attempt for upward gaze, both eyes converge and retract)

21. Enumerate three types of trigeminal neuralgias. (3 marks)

1. Cluster headache
2. Short-lasting, unilateral, neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)
3. Paroxysmal hemicrania or Sjaastad syndrome
4. Primary stabbing headache
5. Raeder paratrigeminal syndrome

22. Describe the clinical features of cluster headache. (3 marks)

- Cluster headache is a type of trigeminal neuralgia
- The typical headache lasts for around 10 minutes to 2 hours and may occur many times a day. Thus, it is called cluster headache
- It is associated with unilateral headache, usually around the temples with features of conjunctival congestion, ptosis, rhinorrhea, epiphora, and miosis
- It may be triggered by alcohol or nitroglycerine ingestion

23. Describe the causes and clinical features of internuclear ophthalmoplegia. (3 marks)

Internuclear Ophthalmoplegia

Causes

Lesions at the medial longitudinal fasciculus (MLF).

Clinical Features

The clinical features are explained considering a left sided lesion (Fig. 8.5.32)

- Straight eyes in primary position
- On attempted right gaze, the right eye abducts with nystagmus, but an inability to adduct the left eye
- The left gaze is normal

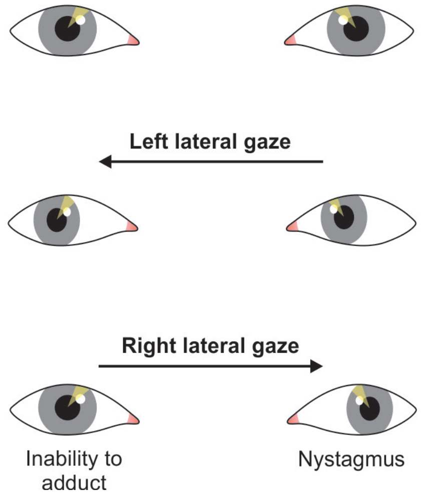


Fig. 8.5.32: Diagrammatic illustration of INO

24. Classify congenital disc anomalies. (3 marks)

Congenital Disc Anomalies

1. **Abnormal size**
    - Hypoplasia
    - Megalopapilla
2. **Abnormal conformation**
    - Morning glory syndrome
    - Tilted disc syndrome
    - Coloboma
    - Optic pit
    - Peripapillary staphyloma
3. **Abnormal tissue**
    - Bergmeister papilla
    - Optic disc drusen
    - Myelinated nerve fiber

SHORT ESSAYS (MISCELLANEOUS)

1. Describe the microscopic layers of the retina and explain the properties of each. (5 marks)

Microscopic Layers of the Retina

The retina has 10 layers

Retinal layer	Properties
Retinal pigment epithelium	• It is composed of a single layer of columnar cells with pigments, resting on the basement membrane – Bruch's membrane.
The layer of rods and cones (outer nuclear layer)	• Rods and cones are photoreceptors that convert light energy into electrical impulses. They are arranged parallel • Rods are abundant in the periphery, whereas cones are abundant in the macula. The fovea is composed of only cones • They are the end organs of vision and are also known as photoreceptors. There are about 120 million rods and 6.5 million cones • Rods contain visual purple (rhodopsin) and are responsible for peripheral vision and vision of low illumination (scotopic vision) • Cones also contain a photosensitive substance (iodopsin) and are primarily responsible for highly discriminatory central vision (photopic vision) and color vision

Contd.

Retinal layer	Properties
External limiting membrane	• It is a fenestrated membrane through which processes of rods and cones pass and come in contact with the RPE
Outer nuclear layer	• This layer contains the nuclei of the rods and cones
Outer plexiform layer	• It contains synapses between the cone pedicles and rod spherules with dendrites of bipolar cells
Inner nuclear layer	• It consists of bipolar cells, amacrine cells, and muller cells. Bipolar cells are first-order neurons of the visual pathway • Amacrine and muller cells have a supportive function
Inner plexiform layer	• It consists of connections between bipolar cells and ganglion cells
Ganglion cell layer	• It contains the nuclei and cell body of the ganglion cells, which form the second-order neurons • They are mainly of two types: midget cells and polysynaptic ganglion cells • Midget cells are in one-to-one correspondence with bipolar cells and are mainly found in the fovea. Polysynaptic cells are mainly in the peripheral retina
Nerve fiber layer	• The nerve fibers arising from the ganglion cells together form the optic nerve
Internal limiting membrane	• It is a basement membrane that separates the vitreous from the retina

2. Describe the surgical anatomy of the vitreous. (5 marks)

Surgical Anatomy of the Vitreous

Vitreous is a gel-like structure that occupies the posterior segment and is responsible for maintaining the structure of the eyeball.

Parts of the Vitreous

1. **Hyaloid membrane**
   - It is the outermost condensation of the vitreous and is divided into an anterior and posterior hyaloid membrane
   - The anterior hyaloid membrane extends from 1.5 mm anterior to the ora serrata circumferentially
   - Various parts of the vitreous are shown in Figure 8.M.1

Part	Description
Wieger's ligament	Firm attachment of the anterior hyaloid membrane to the posterior capsule of the lens
Berger's space	A space in the posterior to the lens and the anterior hyaloid membrane
Patellar fossa	A central 9 mm space in the anterior hyaloid membrane which accommodates the lens
Cloquet's canal	It is a potential canal in the embryological stage which housed the hyaloid artery
Space of Morgagni	It is a space between the optic nerve head and the Cloquet's canal

2. **Cortical vitreous**
   - It is the outer layer of the vitreous that is in contact with the retina posteriorly and the lens and ciliary body anteriorly
   - It is the metabolic center of the vitreous and contains hyalocytes
   - It is around 100 microns in thickness

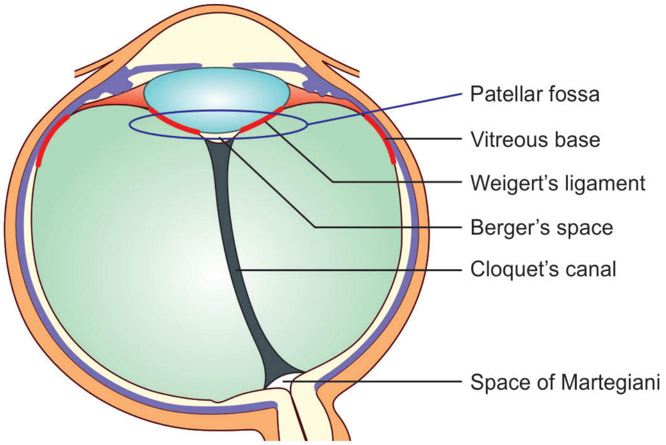


Fig. 8.M.1: Canals and spaces of the vitreous

3. **Core vitreous**
   - Also called medullary vitreous or nucleus
   - It is different from cortical vitreous concerning the presence of hyalocytes and the fact that the fibrils are less loosely woven

Attachments of Vitreous (Fig. 8.M.2)

- It is attached anteriorly at the vitreous base: A 4 mm wide base that straddles the ora serrata
- Thus, it is attached to a 2 mm zone of pars plana and 2 mm to the peripheral retina
- The attachment is so strong that during a traumatic avulsion, the vitreous base along with the ciliary epithelium and retinal epithelium avulse together
- Posteriorly, it is attached around the optic disc, fovea, and along the blood vessels

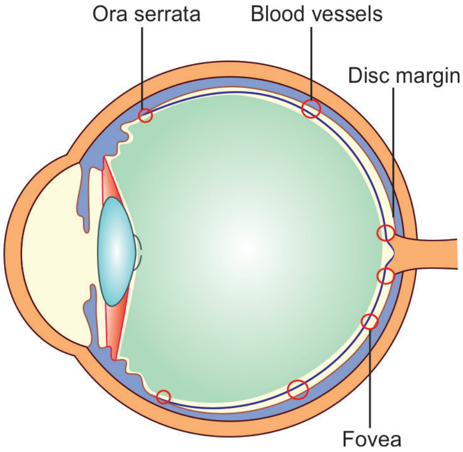


Fig. 8.M.2: Vitreous attachments

3. Describe the clinical features of: (3 marks)

A. Posterior vitreous detachment.

B. Asteroid hyalosis.

C. Synchysis scintillans.

A. Posterior Vitreous Detachment

- It is the detachment of the posterior hyaloid membrane from the optic disc head and is a benign condition
- It is usually seen in old age following synchysis and syneresis
- The synchytic fluid collects between the internal limiting membrane and posterior hyaloid membrane and pushes the posterior aspect of the vitreous forward
- Other causes include:
  1. Following blunt trauma
  2. Intraocular surgery or intravitreal injections

Symptoms

The patient may complain of floaters, many a time described as a spider web, and sometimes associated flashes of light due to a tug felt on the retina during detachment.

Signs

- On examination, a ring of opacity floating anterior to the optic disc is seen (Weiss ring)
- The visual acuity is not affected
- Peripheral retina must be examined to rule out tears

B. Asteroid Hyalosis

- It results due to calcium deposits embedded onto the collagen fibrils gives an asteroid-like appearance
- Usually, there can be associated syneresis

Symptoms

It is usually asymptomatic and is seen in healthy individuals with advanced age.

Signs

White opacities are seen studded in the central vitreous that move when the eyeball moves.

C. Synchysis Scintillans

- It is one of the most spectacular lesions of the vitreous
- Vitreous is laden with small golden crystalline bodies formed of cholesterol
- It is usually seen posttrauma, following resorption of vitreous hemorrhage, post-uveitis

Symptoms

Patients remain asymptomatic.

Signs

Vitreous is also liquefied, as a result, these opacities move every time the eye moves and simulate a shower of golden rain.

4. Enumerate various vitreous opacities and describe their clinical features. (5 marks)

Vitreous Opacities

Vitreous opacity	Clinical features
Muscae volitantes	• Physiological opacities seen in the vitreous • It is more commonly seen in myopes
Persistent hyperplastic primary vitreous (PHPV)	• Also known as persistent fetal vasculature • It results from failure of the primary vitreous structure to regress • It is usually associated with a white pupillary reflex (leukocoria) seen shortly after birth • Other associations are congenital cataracts, glaucoma, microphthalmos, and vitreous hemorrhage
Inflammatory exudates	• Due to vitritis, retinitis, choroiditis, pars planitis, and intermediate uveitis • They are seen as multiple opacities of varying size in the vitreous • They take a long time to recede even after the cause of the inflammation is treated
Tumor cells	• It is seen in primary CNS lymphoma, retinoblastoma • They are seen as large, uniform opacities in the vitreous

Contd.

Vitreous opacity	Clinical features
Asteroid hyalosis	• Calcium deposits embedded onto the collagen fibrils gives an asteroid-like appearance • Usually, there can be associated syneresis • It is usually asymptomatic and is seen in healthy individuals with advanced age • Usually needs no treatment
Synchysis scintillans	• It is one of the most spectacular lesions of the vitreous • Vitreous is laden with small golden crystalline bodies formed of cholesterol • It is usually seen post-trauma, following resorption of vitreous hemorrhage, post uveitis • Vitreous is also liquefied, as a result, these opacities move every time the eye moves and simulate a shower of golden rain
Amyloidosis	• Amyloid material is deposited in the vitreous as a part of generalized amyloidosis • These vitreous opacities appear linear with attachments to the retina and the posterior lens surface

5. Describe the etiology, clinical features, and management of vitreous hemorrhage. (2+1+2=5 marks)

Vitreous Hemorrhage

Vitreous hemorrhage is bleeding into the vitreous cavity.

Etiology

- Trauma—either blunt or penetrating in nature
- Retinal vascular proliferative disorders like proliferative diabetic retinopathy, venous occlusions, Eales' disease, etc.
- During posterior vitreous detachment
- Following inflammation of uvea—pars planitis, chorioretinitis
- Blood dyscrasias like leukemia, sickle cell retinopathy, hemophilias, scurvy
- A choroidal neovascular membrane may occasionally bleed
- Neoplasms: Vitreous hemorrhage may occur due to erosion of vessels due to acute necrosis in tumors like retinoblastoma

Clinical Features

Symptoms

- It depends on the amount of bleed. If it is a small bleed, it presents as floaters
- Massive bleed can obscure vision to an extent of just perception of light
- It is a cause for sudden painless loss of vision

Signs

- The anterior segment may show signs of associated ocular trauma or new vessels on the iris suggestive of vascular proliferation
- It is diagnosed by ophthalmoscopy. On distant direct ophthalmoscopy, a dark glow is seen massive bleed. In cases of small hemorrhages, floating opacities can be seen
- Indirect ophthalmoscopy should be done to find the source of bleed if there is visibility
- B scan is mandatory if fundus cannot be visualized to rule out retinal detachment if clinical features point towards it. B scan shows moderate echogenic ill-defined vitreous opacities, which move along with the movement of the eye. If the hemorrhage is an old one, the echogenicity increases

Management

- If uncomplicated, the patient should be advised absolute bed rest in a propped-up position so that bleed settles down
- Once the medium has cleared, look for the source of the bleed and treat it by LASER photocoagulation
- Vitrectomy is advised in the following circumstances
  1. Unresolved hemorrhage for more than 3 months
  2. Associated retinal detachment
  3. The other eye is blind
  4. Loss of vision in the other due to the same cause

6. Describe the clinical presentation of PHPV. (5 marks)

The persistent hyperplastic primary vitreous is now known as persistent fetal vasculature and occurs due to a failure of the primary vitreous to regress.

Symptoms

- It is usually unilateral and seen in infants
- Parents bring the child to the ophthalmologist with a history of seeing white pupillary reflex (leukocoria)

Signs

Persistent fetal vasculature can occur in anterior or posterior forms. Most of the cases are associated with microphthalmos.

Anterior PHPV

- Anterior PHPV is usually associated with a shallow anterior chamber, Mittendorf dot, congenital cataract (leukocoria), long ciliary processes, congenital glaucoma
- It is usually associated with microphthalmos as the normal development of the eye is arrested
- It can be treated with lensectomy with the removal of the fibrovascular retrolental membrane
- Visual prognosis remains poor due to the development of glaucoma and amblyopia

Posterior PHPV

- The anterior chamber is normal, and the lens is usually clear
- A stalk of tissue is seen emanating from the center of the optic disc and traveling towards the lens and fanning outwards the anterior retina
- It is also associated with Bergmeister papilla (tuft of vessels and surrounding gliosis at the disc surface), the persistent hyaloid artery leading to recurrent hemorrhages in the vitreous, retinal folds, and tractional retinal detachments
- The visual prognosis is usually poor

7. Describe the indications and methods of vitrectomy. (5 marks)

Vitrectomy

Indications

1. **Inflammatory causes**
   - Endophthalmitis
   - Intermediate uveitis not responding to medications
   - Cystoid macular edema

2. **Trauma/iatrogenic**
   - Traumatic retail dialysis, detachment
   - Macular hole
   - Nucleus drop in vitreous
   - Intravitreal foreign body
   - Posteriorly dislocated lens
3. **Degenerative causes**
   - Retinal detachment
   - Epiretinal membrane
4. **Proliferative causes**
   - Proliferative diabetic retinopathy with complications (retinal detachment)
   - Proliferative vitreoretinopathy
   - Proliferative retinopathy due to any cause
5. **Vitreous hemorrhage**

Methods

1. **Anterior vitrectomy**
   - It refers to the removal of the anterior part of the vitreous
   - It is usually done during a posterior capsular rent during cataract extraction
2. **Core vitrectomy**
   - Removal of the central bulk of the vitreous
   - It is usually indicated in endophthalmitis
3. **Subtotal and total vitrectomy**
   - In this, almost the whole of the vitreous is removed
4. **Open sky vitrectomy**
   - Open sky vitrectomy is performed through the primary wound using a vitrector
   - If the vitrectomy machine is unavailable, it can be performed using de Wecker's scissors (sponge vitrectomy)
5. **Pars plana vitrectomy**
   - Three port technique: Three separate incisions are given in the pars plana region, depending on the guage of the vitrector
   - The cutting and aspiration functions are contained in one probe, illumination is provided by a separate fiberoptic probe, and an infusion is provided by a cannula introduced through the third pars plana incision

SHORT ANSWERS (MISCELLANEOUS)

1. Describe the fate of vitreous hemorrhage. (3 marks)

Fate of Vitreous Hemorrhage

- Complete absorption of hemorrhage , with clear vitreous, in about 6 weeks
- Organized hemorrhage in the form of yellowish brown membrane behind the lens (Ochre membrane)
- Synchysis, red cell degeneration and ghost cell glaucoma, especially in aphakics
- Fibrovascular proliferation leading to tractional retinal detachment

2. Explain the management of vitreous hemorrhage. (3 marks)

- If the vitreous hemorrhage is uncomplicated, the patient should be advised absolute bed rest in propped position so that bleed settles down

- Once the medium has cleared, look for the source of the bleed and treat it by LASER photocoagulation
- Vitrectomy is advised in the following circumstances
  1. Unresolved hemorrhage for more than 3 months
  2. Associated retinal detachment
  3. The other eye is blind
  4. Loss of vision in the other due to the same cause

3. Explain the B-scan characteristics of vitreous hemorrhage. (3 marks)

- B scan shows multiple mobile point echos with moderate echogenicity in the vitreous cavity
- If the hemorrhage is an old one, the echogenicity increases
- B-scan can also identify associated retinal or vitreous detachment

4. Describe the development of vitreous. (3 marks)

The development of vitreous can be studied in the following steps.

Primary Vitreous

- The primary vitreous is mesenchymal in origin and is a vascular structure (Fig. 8.M.3)
- In the fetus, the blood supply of the eye is mainly through the hyaloid artery which is a branch of the internal carotid artery that enters the optic stalk to supply the lens and rest of the eye
- There is no venous structure, venous blood drains into the surrounding choroidal veins
- With the development of the retinal vessels that are triggered by VEGF (angiogenesis), the hyaloid artery starts regressing from its midpoint and proceeds anteriorly and posteriorly
- The lens is then nourished by aqueous once the hyaloid system of vessels regresses

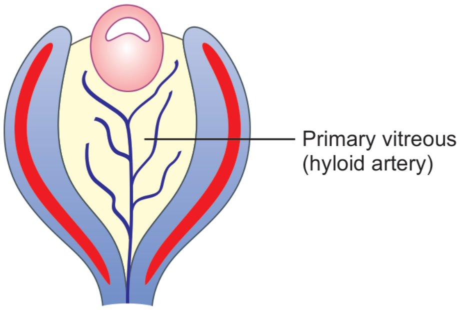


Fig. 8.M.3: Primary vitreous

Secondary Vitreous

The secondary vitreous is neuroectodermal in origin as it is produced by the optic cup (Fig. 8.M.4).

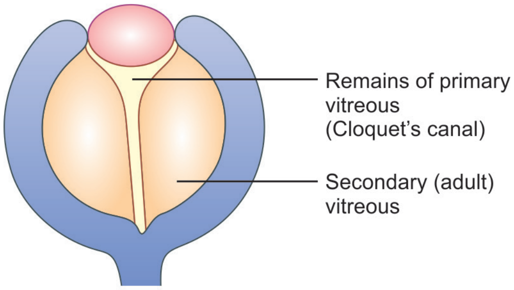


Fig. 8.M.4: Formation of secondary vitreous

Tertiary Vitreous

Tertiary vitreous is represented by ciliary zonules (Fig. 8.M.5).

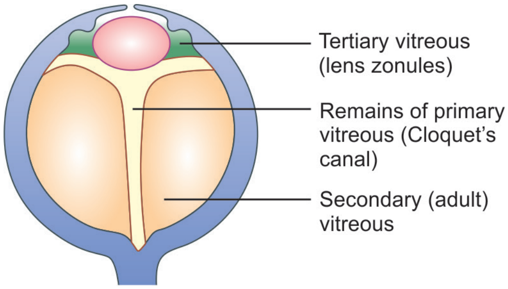

Tertiary vitreous (lens zonules)
Remains of primary vitreous (Cloquet's canal)
Secondary (adult) vitreous

Fig. 8.M.5: Tertiary vitreous

5. Enumerate various vitreous substitutes. (3 marks)

- Following the vitrectomy, the vitreous space should be substituted to maintain the intraocular pressure and globe integrity
- These substances are called vitreous substitutes

Gases	Liquids
Sulfur hexafluoride • Lasts for 10 days	Ringer lactate or balanced salt solution
Perfluoropropane • Lasts for 28 days	Perfluorocarbon liquids • Mainly used to cover giant tears, during epiretinal peeling to keep tamponade over the macula, to remove a foreign body or dropped nucleus
Air • In uncomplicated cases, gets absorbed within 3 days	Silicone oil • For retinal detachment surgeries

CHAPTER

9

Miscellaneous

COMPETENCY

OP9.1 DEMONSTRATE THE CORRECT TECHNIQUE TO EXAMINE EXTRA OCULAR MOVEMENTS (UNIOCULAR & BINOCULAR)

SHORT ESSAYS

1. Classify extraocular movements and state the extraocular muscles responsible for each cardinal position. (3+2 marks)

Extraocular movements are classified as:

1. Monocular (ductions) movements
2. Binocular (versions and vergences) movements

Ductions/uniocular movements	• Adduction: Medial rotation along the vertical axis • Abduction: Lateral rotation along the vertical axis • Supraduction: Upward rotation along the horizontal axis • Infraduction: Downward rotation along the horizontal axis • Incycloduction (intorsion): Inward rotatory movement along the anteroposterior axis • Excycloduction (extorsion): Outward rotatory movement along the anteroposterior axis
Versions/binocular symmetric movements	• Dextroversion: Movement of both eyes to the right • Levoversion: Movement of both eyes to left • Supraversion: Movement of both eyes upwards • Infraversion: Movement of both eyes downwards • Dextrocycloversion: Rotational movement around the anteroposterior axis, in which the superior pole of the cornea of both the eyes tilts towards the right • Levocycloversion: Rotational movement around the anteroposterior axis, in which the superior pole of the cornea of both the eyes tilts towards the left
Vergences/bilateral Asymmetric movements	• Convergence: Both eyes move towards the nose along the vertical axis • Divergence: Both eyes move outwards along the vertical axis

Cardinal Positions of Gaze

1. **Dextroelevation:** Right superior rectus, left inferior oblique
2. **Dextroversion:** Right lateral rectus, left medial rectus
3. **Dextrodepression:** Right inferior rectus and left superior oblique
4. **Levoelevation:** Left superior rectus, right inferior oblique

5. **Levoversion:** Left Lateral rectus, right medial rectus
6. **Levodepression:** Left inferior rectus, right superior oblique

2. Describe the origin, insertion, nerve supply, and actions of all the extraocular muscles. (1+1+1+2 marks)

Origin, Insertion, Nerve Supply

Muscle	Origin	Insertion	Nerve supply
Superior rectus	Annulus of Zinn	7.7 mm behind superior limbus, 23° with the optical axis	Oculomotor (superior division)
Inferior rectus		6.5 mm behind inferior limbus, 23° with the optical axis	Oculomotor (inferior division)
Medial recuts		5.5 mm behind medial limbus, parallel to the optical axis	Oculomotor (superior division)
Lateral rectus		6.9 mm behind lateral limbus, parallel to the optical axis	Abducens
Superior oblique	Superior and medial to the optic foramen	The upper and outer part of the sclera behind the equator, 51° with the optical axis	Trochlear
Inferior oblique	The orbital part of maxilla-lateral to the orifice of NL duct	The lower and outer part of the sclera behind the equator, 51° with the optical axis	Oculomotor (inferior division)

Actions

Muscle	Primary action	Secondary action	Tertiary action
Superior rectus	Elevation (best when abducted by 23°)	Intorsion	Adduction
Inferior rectus	Depression (best when abducted by 23°)	Extorsion	Adduction
Medial recuts	Adduction		
Lateral rectus	Abduction		
Superior oblique	Intorsion	Depression (best when adducted 51°)	Abduction
Inferior oblique	Extorsion	Elevation (best when adducted 51°)	Abduction

3. Describe the abnormalities of binocular singular vision. (5 marks)

Anomalies of Binocular Single Vision

1. **Suppression**
   - This is a phenomenon characterized by temporary active cortical inhibition of images from the squinting eye
   - It is seen when both eyes are open
   - It is due to two different objects projected onto the visual cortex from two eyes
   - Clinically suppression can be tested using the Worth 4-dot test, 4PD base out prism test, and synoptophore
2. **Abnormal retinal correspondence**
   - Normal retinal correspondence is a state characterized by a precise physiological relationship between the corresponding points of the two retinae, such that when an object is projected onto these two points, it produces a single image

- NRC is broken when the visual axes from both eyes are not parallel
    - Confusion (Fig. 9.1.1)
        - o It is characterized by the overlapping of two images formed in each eye, alternatively as exemplified in this picture (stimulation of two foveal points by different images)
    - Diplopia (Fig. 9.1.2)
        - o It is characterized by the formation of two images of the same object due to stimulation of two different points on the retina (one foveal, one extrafoveal) due to malalignment of visual axes

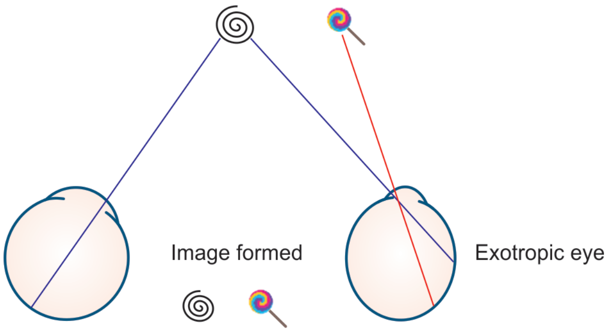


Fig. 9.1.1: Confusion

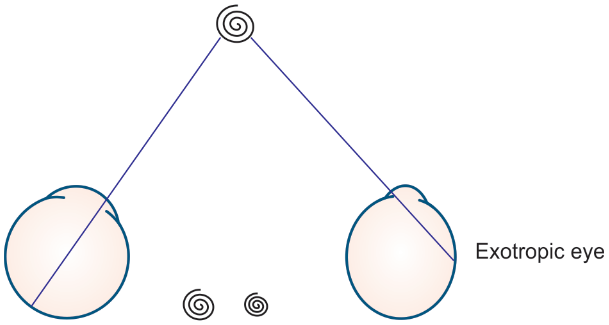


Fig. 9.1.2: Diplopia

3. **Amblyopia**

- It occurs due to long standing loss of normal retinal correspondence due to many factors
- It is defined as "partial or complete, unilateral or bilateral loss of visual acuity in the absence of pathology of ocular media, retina, and visual pathway. It occurs at the cortical level when the image formed by the affected eye is either blurred or of a different size
- It occurs in the age group from birth to 6 years, which is crucial for the development of binocular single vision

SHORT ANSWERS

1. Define yoke muscles and name two pairs of yoke muscles. (1+2 marks)

- Yoke muscles are pairs of muscles involved in the same version
- They are also called contralateral synergists
    1. Right superior rectus, left inferior oblique
    2. Right lateral rectus, left medial rectus
    3. Right inferior rectus and left superior oblique
    4. Left superior rectus, right inferior oblique
    5. Left lateral rectus, right medial rectus
    6. Left inferior rectus, right superior oblique

2. Describe the laws of extraocular motility. (3 marks)

Laws of Extraocular Motility

1. **Hering's law of equal innervation**

- Equal and simultaneous innervation flows from the brain to a pair of yoke muscles or muscles involved in the same binocular movement

- For example,
  1. *During dextroversion:* Right LR and left MR muscles
  2. *During convergence:* Both MR
  3. *During dextroelevation:* Right SR and left IO

2. **Sherrington's law of reciprocal innervation**
   - During a binocular movement, there is an increased flow of innervation to the contracting muscle and decreased flow of innervation to the relaxing antagonist muscle
   - For example, during dextroversion: an increased innervation flow to the right LR and left MR is accompanied by decreased flow to the right MR and left LR muscles

COMPETENCY

OP9.2 CLASSIFY, ENUMERATE THE TYPES, METHODS OF DIAGNOSIS AND INDICATIONS FOR REFERRAL IN A PATIENT WITH HETEROTROPIA/STRABISMUS

SHORT ESSAYS

1. Define strabismus. How is it classified? (1+4 marks)

Strabismus/Squint

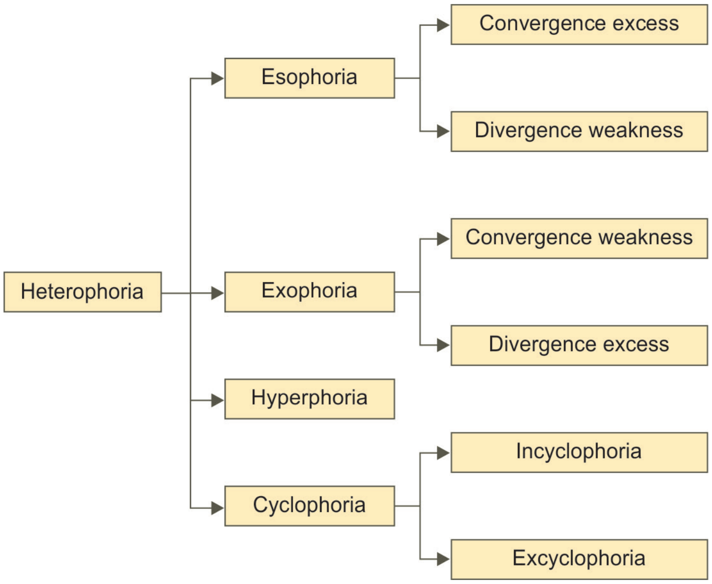
Definition

When the visual axes of both eyes are not parallel

Classification

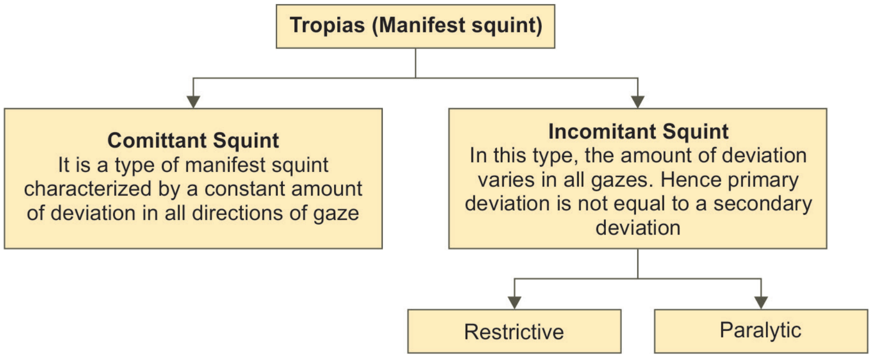
Strabismus is classified
1. Latent squint (phoria)
2. Manifest squint (tropia)

1. **Classification of phoria**
   - The eyes are kept aligned by fusion, when fusion is broken by covering an eye, squint manifests


Heterophoria
Esophoria
Convergence excess
Divergence weakness
Exophoria
Convergence weakness
Divergence excess
Hyperphoria
Cyclophoria
Incyclophoria
Excyclophoria

2. **Classification of tropia**

- Another way of classification of squint is based on the direction of squint
    - Esotropia
    - Exotropia
    - Hypertropia
    - Hpotropia


Tropias (Manifest squint)
Comittant Squint
It is a type of manifest squint characterized by a constant amount of deviation in all directions of gaze
Incomitant Squint
In this type, the amount of deviation varies in all gazes. Hence primary deviation is not equal to a secondary deviation
Restrictive
Paralytic

2. Contrast comitant and incomitant squint. (5 marks)

Feature	Concomitant squint	Incomitant squint
Cause	Usually congenital Decreased vision may be present in either eye	Acquired
Deviation	Same in all gazes	Changes in all gazes The secondary deviation is greater than primary in paralytic strabismus The secondary deviation is lesser than primary on restrictive strabismus
Symptoms	No diplopia No nausea/vomiting	Diplopia Nausea and vomiting induced by diplopia
Restriction of movements	No	Yes
Abnormal head posture	No	Yes
Amblyopia/suppression/ARC	Yes, can be there	Not necessarily
Past pointing	No	Present

3. Describe the evaluation of a case of squint in the pediatric age group. (5 marks)

Every child with a squint should undergo a complete ophthalmic examination including visual acuity, anterior segment evaluation, pupil, and fundus.

Tests to Differentiate Tropia from Phoria

This is done by using a cover test and an alternate cover test

- The cover-uncover test is performed by occluding one eye and observing its behavior while uncovering. If the eye moves inside during cover it is esophoria, if it moves outside, it is exophoria and likewise
- Alternate cover test
    - It is used to break fusion

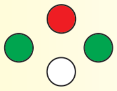
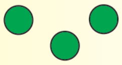
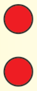
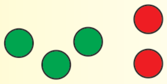
- One eye is occluded for several seconds with the other eye focusing on a distant object
- Then the occluder is moved to another eye, back and forth several times to break fusion
- In the case of a phoria, squint will manifest

Tests Performed for Both Comitant and Incomitant Squint

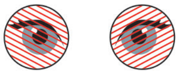
1. **Hirschberg test**
    - Here patient is asked to focus on a distant source of light and corneal reflex is observed
    - In orthophoric condition, both corneal reflexes must be at the center of the pupil
    - In phorias, this is maintained by fusion, hence may not be able to ascertain the amount of squint
    - Depending on the position of the corneal reflex, the amount of squint can be roughly estimated (1° = 2 Prism diopters)
2. **Prism bar cover test**
    - Used to calculate the amount of deviation using prisms
    - Vertical and horizontal prism bars are available for respective types of squint
    - After cover-uncover, the type of deviation is identified
    - With one eye occluded, a prism bar is placed in front of the other with an apex towards the deviation
    - Its strength is then progressively increased until there is no movement of the eye undercover
3. **Testing extraocular movements**
    - It is tested by showing an object of interest to the child and observing the movements of the eye when the object is moved in an 'H'-shaped manner
    - Uniocular and binocular movements should be tested
    - It is followed by tests for convergence

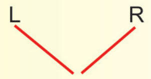
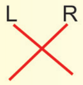
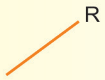
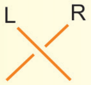
Tests for a Comitant Squint

1. **Worth's four-dot test**
    - It is a test for binocular sensory function. Red glasses are placed in front of the right eye and green in front of the left eye
    - The patient is asked to fixate on a screen with four circles: Two green, one red, and one white
    - The results are tabulated below:

Normal – BSV, abnormal retinal correspondence if there is squint	
Right suppression	
Left suppression	
Alternating suppression Diplopia	

2. **Bagolini's striated glasses**
   - In this, striated glasses are placed in the manner showed in the picture
   - The patient is asked to look at a source of light
   - The results are tabulated below:



Normal, harmonious ARC	
Diplopia	
Left suppression	
Central scotoma as in microtropia	

3. **Synoptophore**
   - It is the equipment used to test levels of binocularity
   - It also helps in assessing the angle of deviation
4. **Tests for stereopsis**
   - Titmus fly test and TNO tests are available for testing the degree of stereopsis (depth perception)

Tests for an Incomitant Squint

1. **Diplopia charting**
   - Subjective test for recording the motility of the eyes using a beam of light and red-green glasses
   - The light is projected in 9 quadrants and the patient is asked to report if he seen splitting of lines
2. **Hess charting**
   - Using an electronic chart where points of red light are projected on an electronic screen
   - The patient is given a green pointer and asked to join the red points displayed on the screen
   - It is an objective test for identifying diplopia and paralytic strabismus
3. **Forced duction test**
   - It is done under general anesthesia, as touching extraocular muscles can lead to oculocardiac reflex
   - The muscle in suspicion is held with forceps and moved in the direction of its action. Inability to move freely points to a paralytic cause
   - Mechanical restriction is seen in restrictive strabismus

4. Describe the clinical features and management of congenital esotropia syndrome. (3+2 marks)

Congenital Esotropia Syndrome

Also called essential esotropia.

Clinical Features

Symptoms

- Usually presents before 6 months of age
- Brought by parents with a history of squint

Signs

- Constant, large (about 30°) of esotropia
- Often associated with alternate fixation, latent nystagmus, and inferior oblique overaction
- The child uses the right eye in the left gaze and the left eye in the right gaze (cross fixation)
- There is amblyopia in about 20–40% of patients
- Due to this, it may be confused with bilateral lateral rectus palsy. It can be demonstrated by a doll's eye maneuver or unilateral eye patching
- There is usually no associated neurological abnormality

Management

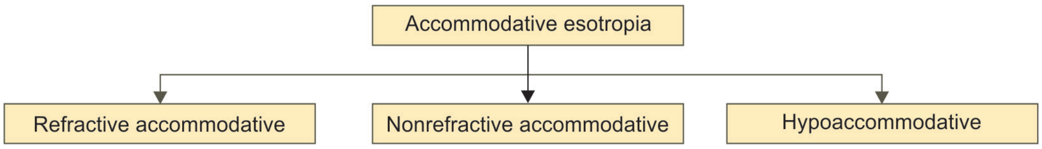
- Surgical correction should be done before the onset of amblyopia, mostly by 12 months
- It consists of bilateral medial rectus recession (weakening) or unilateral medial rectus recession with lateral rectus resection (strengthening)
- If associated with inferior oblique overaction, that should be addressed too
- Initial correction of refractive error and amblyopia should be done before squint surgery

5. Describe the types and clinical features of accommodative esotropia. (5 marks)

Accommodative Esotropia

- Accommodative esotropia is a type of comitant squint in which the esotropia is due to excess accommodation
- It occurs as a result of overactive convergence associated with accommodation reflex

Types



Clinical Features

Refractive accommodative	• It is characterized by high uncorrected hypermetropia around +4 to +8D • It usually presents at the age of 2–4 years • It is fully correctable by proving correction for hypermetropia • May be associated with amblyopia if not corrected
Non-refractive accommodative	• The hypermetropia is not as large as the first type • It is due to a high AC/A ratio (increased convergence associated with normal accommodation) • These respond to bifocal glasses with the addition of +3D for near vision
Hypoaccommodative	• Convergence is high due to less accommodation • They are treated by correction of hypermetropia and addition of +3D for near

6. Describe the clinical features of an isolated one-sided oculomotor palsy. (5 marks)

Clinical Features of Oculomotor Palsy

An isolated ipsilateral oculomotor palsy is characterized by the following:
- The eye is "down and out", abducted and depressed, when an effort is made to depress the eyeball, the eye intorts (due to the action of superior oblique). Limitation of all movement except abduction and intorsion
- Ptosis: Due to weakness of the levator muscle, may range from mild to severe
- Abnormal head posture: Chin elevation and face turn to the opposite side.
- Loss of pupillary light and near reflex: results in anisocoria

Special Features to Localize the Level of Lesion

Bilateral ptosis with contralateral superior rectus palsy	Nuclear level
Sparing of pupillary reflex	Medical/ischemic causes of oculomotor nerve palsy
Associated with multiple cranial nerves like trochlear, trigeminal, and abducens nerve	Cavernous sinus Orbital apex
Associated with headache	PICA aneurysm and subarachnoid hemorrhage Raised intracranial pressure (Hutchinson's pupil)

7. Describe the principles of management of squint. (5 marks)

Principles of Management of Squint

1. **Recognition of the type of squint and assessment of binocularity**
    - Squint requires a battery of tests that can be time and painstaking for the examiner as well as the patient
    - It consists of cover-uncover tests, Krimsky's test, and tests for binocular function like Worth's four dot test, Bagolini's striated glasses
    - It is followed by tests for extraocular motility
    - At the end of the examination, one must ascertain the etiology, type, degree, and possible complications of squint
2. **Type of therapy—medical, orthoptic, or surgical**
    - Goals for treatment of strabismus
        - Restore binocularity
        - Bring about ocular alignment

    - Alleviate abnormal head posture
    - Improve aesthetics
  - Some types of squint are treated by treating the refractive error (accommodative esotropia)
  - Medical treatment is indicated in reversible causes of squint
    - Correction of blood sugars in diabetes
    - Use of botulinum toxin in a restrictive squint
    - Neostigmine for myasthenia gravis
  - Surgical treatment
    - Includes muscle-weakening procedures in cases of overaction of muscles (recession, disinsertion), and strengthening procedures like resection for weak muscles
    - These procedures are helpful in an incomitant squint. 1 mm of resection or recession of the medial rectus corrects around 5PD of deviation and 1mm of resection or recession of the lateral rectus corrects 3PD of deviation
    - In paralytic squint, strengthening procedures are not very useful. They are treated with muscle transposition. Other muscles are attached to the insertion of the weak muscle

3. **Treatment of associated pathologies**
   - Amblyopia: It is important to prevent strabismic amblyopia and thus must be corrected before the surgical correction of quint
   - Refractive error
   - Other ocular abnormalities: Removal of the offending agent that primarily caused the squint like congenital cataract or corneal opacity
   - Associated systemic disorders like removal of an intracranial mass

8. Describe the ocular features of myasthenia gravis. (5 marks)

- Myasthenia gravis is an autoimmune disorder affecting the neuromuscular junction of skeletal muscles
- It is characterized by increased immunoglobulin-mediated destruction of acetylcholine receptors at the neuromuscular junction of voluntary muscles
- This leads to decreased conduction of stimulus across the neuromuscular junction leading to muscle fatigue
- It affects females more and can be ocular, systemic, or generalized
- Ocular myasthenia is a subtype of MG characterized by ocular features only, without a systemic manifestation

Ocular Features

1. **Ptosis**
   - It is the first presentation as these muscles have the highest rate of firing in the whole body (fast-twitch muscles)
   - Ptosis increases on prolonged upgaze
   - Cogan's lid twitch is elicited by asking the patient to look down for 1 minute and then suddenly elevate the lids
   - An upshot of lids is seen at the beginning of the saccade
2. **Diplopia**
   - Variable diplopia
   - More towards the end of the day
   - The restricted extraocular movements do not follow any particular pattern

3. **Pupillary reactions**
   - Normal
4. **Positive ice test**
   - Increase in neuromuscular transmission with cold
   - Ice application improves ptosis

9. Describe the uses of a synoptophore. (5 marks)

The synoptophore is also known as the major anomaloscope.

Uses

1. **Estimation of grades of binocularity (Fig. 9.2.1)**
   - Conventionally, there are three grades of binocular singular vision and they are measured using a synaptophore:
     i. *Grade I: Simultaneous Macular Perception*
       - Both eyes can see two dissimilar objects simultaneously
       - When two dissimilar objects are presented to both eyes through a synaptophore, there are fused
       - For example, when a cage is shown to the right eye and a parrot in the left eye, the binocular system perceives it as a bird in the cage

     ii. *Grade II: Fusion*
       - The brain can fuse two incomplete similar objects to create the image of a single object. It has two components: sensory and motor
       - When two partial images are presented to each eye at the same time through a synaptophore, they are fused to form an image
       - For example, when a rabbit is projected to the right eye and hands holding a bouquet in the left eye, they are seen as a rabbit holding a bouquet

     iii. *Grade III: Stereopsis*
       - It is the most advanced form of binocular vision and it is the ability to perceive the third dimension i.e., depth perception.
2. **Detection of abnormal retinal correspondence**
   - It is done by determining the subjective and objective angles of the squint
   - If the two angles are equal, then there is normal retinal correspondence
   - If the objective angle is greater than the subjective, then it is abnormal retinal correspondence. The difference is called the angle of anomaly

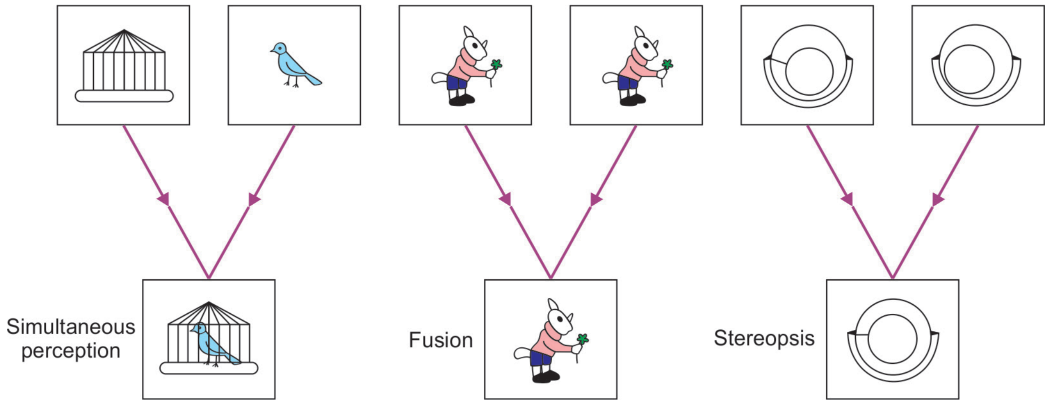


Fig. 9.2.1: Grades of binocularity

10. Describe the clinical features of paralytic strabismus. (5 marks)

Clinical Features of Paralytic Strabismus

1. **Deviation**
   - The deviation of the affected eye is away from the direction of action of the muscle
   - In lateral rectus palsy, the eye is in the adducted position
   - When the sound eye is fixing, the deviation of the affected eye is called the primary deviation
   - When the affected eye is made to fix, the deviation of the eye undercover is called secondary deviation
   - It is always greater than the primary deviation in paralytic strabismus
   - The amount of deviation is measured in each of the nine gazes and finally for distance and near
2. **Restricted ocular movements**
   - Movements are restricted in the direction of paralytic muscle
3. **Abnormal head posture**
   - It is adopted to avoid diplopia
   - Common head postures are:
     i. Face turn to opposite side: Lateral rectus palsy
     ii. Head tilt to the opposite side, chin depression and face turn to the opposite side in fourth nerve palsy
4. **Past pointing**
   - It is due to increased innervation reaching a muscle due to which the patient has a false impression of the object being farther away than its normal position

SHORT ANSWERS

1. Enumerate three causes for pseudostrabismus. (3 marks)

1. **Pseudo-esotropia:** Telecanthus (broad nasal bridge), epicanthal fold, high myopia (negative angle kappa)
2. **Pseudo exotropia:** Hypertelorism, high hypermetropia(positive angle kappa)
3. **Pseudo-vertical squint:** Ptosis or lid retraction

2. Describe the procedure of Hirschberg's test. (3 marks)

Hirschberg's Test

Procedure

- The Hirschberg test is performed as a preliminary test to gauge the amount of squint
- The patient is asked to focus on a distant source of light and corneal reflex is observed
- In an orthophoric condition, both corneal reflexes must be at the center of the pupil (Fig. 9.2.2)
  - If the reflex falls on the nasal side of the center of the cornea, then it is exotropia
  - If the reflex falls on the temporal aspect of the cornea, then it is esotropia
  - If the reflex is above or below the center of the cornea, then it is hypertropia and hypotropia respectively
- In phorias, this is maintained by fusion, hence may not be able to ascertain the amount of squint
- Depending on the position of the corneal reflex, the degree of squint can be estimated (1° = 2 prism diopters)

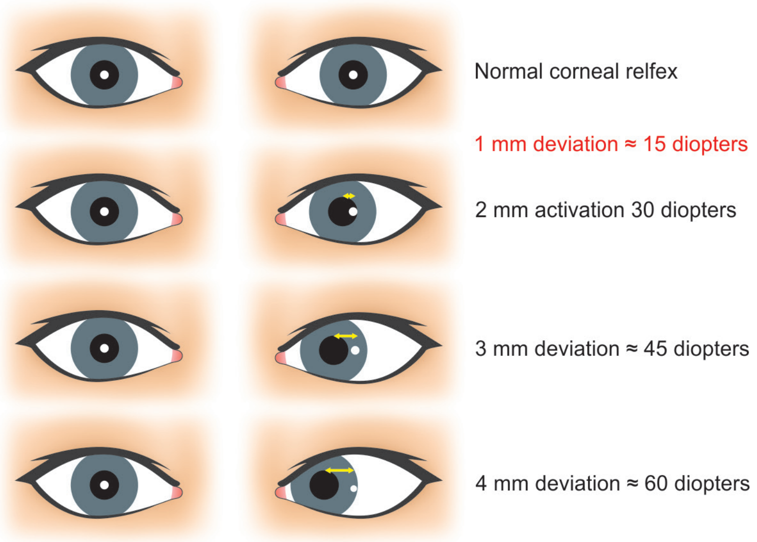

Normal corneal relfex
1 mm deviation ≈ 15 diopters
2 mm activation 30 diopters
3 mm deviation ≈ 45 diopters
4 mm deviation ≈ 60 diopters

Fig. 9.2.2: Results of Hirschberg's test

3. What is the AC/A ratio? Describe its importance. (3 marks)

AC/A Ratio

- AC/A ratio describes the amount of accommodative convergence that happens per amount of accommodation
- It is normally 4–6 PD of convergence per diopter of accommodation

Importance

- AC/A ratio is an important calculation in the case of squint
- Increased AC/A ratio signifies exuberant convergence and thus results in non-refractive accommodative esotropia

4. Describe the features of left-sided fourth nerve palsy. (3 marks)

Left-sided Fourth Nerve Palsy

- The patient presents with sudden onset vertical diplopia
- There is a head tilt to the right side with a depressed chin
- In the primary position, there is left hypertropia, which worsens on the right gaze and better on the right-sided head tilt (remembered as BOOT: better on opposite tilt)
- On attempted downgaze, the left eye cannot depress due to loss of action of superior oblique, whose action is depression. As a result, the hypertropia increases on downgaze and so does the diplopia (WOOG: worse on opposite gaze)
- It is diagnosed based on Park's three-step test and Beilchowsky's test

5. Describe the features of acute right abducens palsy. (3 marks)

- Right abducens palsy results in the palsy of the right lateral rectus
- The amount of esotropia depends on the degree of palsy. That is, in severe palsy, the degree of squint is higher

- The type of diplopia is horizontal and is higher in the direction of gaze towards the side of the palsy
- There is a face turn to the left
- In right abducens palsy, there is worsening of horizontal diplopia when there is an attempted right gaze as the left eye moves towards the right, but the right eye cannot abduct as a result of palsy

6. Describe the procedure of diplopia charting. (3 marks)

- Diplopia charting is a simple bedside subjective test to understand paralytic/restrictive squint
- This is done using red-green glasses, with red color in front of the right eye and left in front of the left eye
- The examiner stands behind the patient, who is seated in a dark room facing a wall at least 4 feet away. Using a slit of the ophthalmoscope or a retinoscope, a beam of light is projected on the wall, and the patient is asked to describe it in all nine gazes
- Normally, one line-half green and half red, is seen. In presence of diplopia, two lights are seen, away from each other. The quadrant of the highest separation should be noted
- For example, left lateral rectus palsy (Fig. 9.2.3)

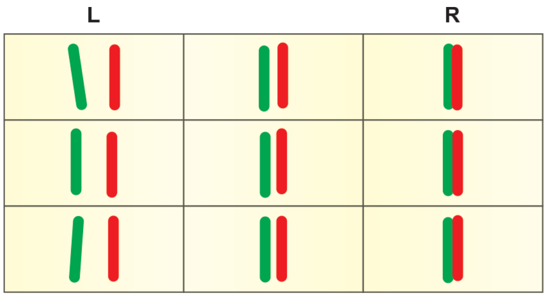


Fig.9.2.3: diplopia charting in a left lateral rectus palsy

7. Describe the clinical features of Duane's retraction syndrome. (3 marks)

- Duane's syndrome is a type of congenital cranial dysinnervation disorder
- It occurs due to paradoxical innervation of the medial rectus or lateral rectus or both
- The characteristic feature is a restriction of abduction or adduction or both
- Also, there is a retraction of the globe and a narrowing of the palpebral fissure on attempted abduction
- The eyes may be orthophoric, esotropic, or exotropia in primary position

Type I DRS	Limitation of abduction
Type II DRS	Limitation of adduction
Type III DRS	Limitation of both abduction and adduction

8. Describe the clinical features of double elevator palsy. (3 marks)

- Double elevator palsy is a congenital condition characterized by supranuclear palsy of the superior rectus and inferior oblique of the involved eye
- There is a unilateral limitation of upgaze with hypotropia of the affected side
- The limitation is the same in adduction and abduction

9. Describe the method of performing Krimsky's prisms bar test. (3 marks)

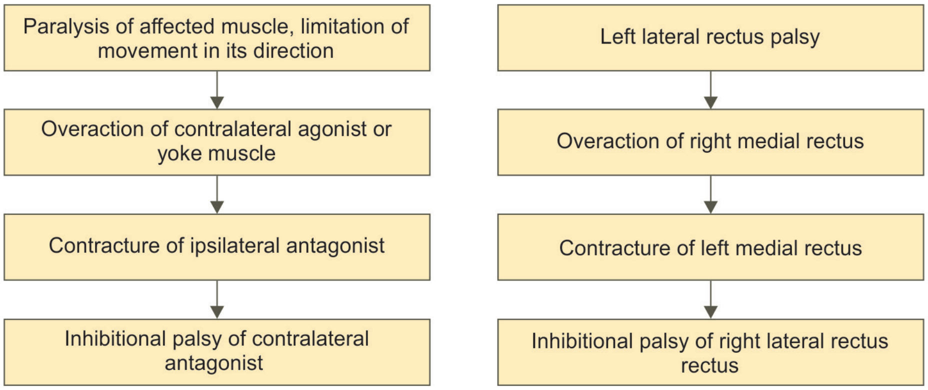
- Krismky's prism bar test is the standard clinical method to calculate the amount of ocular deviation
- Vertical and horizontal prism bars are available for respective types of squint

- After cover-uncover, we know which is the type of deviation
- With one eye occluded, a prism bar is placed in front of the other with the apex towards the deviation
- Its strength is then progressively increased until there is no movement of the eye undercover

10. Enumerate myogenic causes for squint. (3 marks)

1. **Congenital causes:** Muscle hypoplasia, malinsertion, weakness
2. **Traumatic lesions:** Disinsertion, laceration, incarceration of muscle into fracture segment
3. **Inflammatory lesions:** Like myositis, pseudotumor
4. **Myopathies:** Thyroid orbitopathy, chronic progressive external ophthalmoplegia

11. Describe the pathological sequelae of a left abducens palsy. (3 marks)


Paralysis of affected muscle, limitation of movement in its direction
Overaction of contralateral agonist or yoke muscle
Contracture of ipsilateral antagonist
Inhibitional palsy of contralateral antagonist
Left lateral rectus palsy
Overaction of right medial rectus
Contracture of left medial rectus
Inhibitional palsy of right lateral rectus rectus

12. Write the similarities and dissimilarities between paralytic and restrictive strabismus. (3 marks)

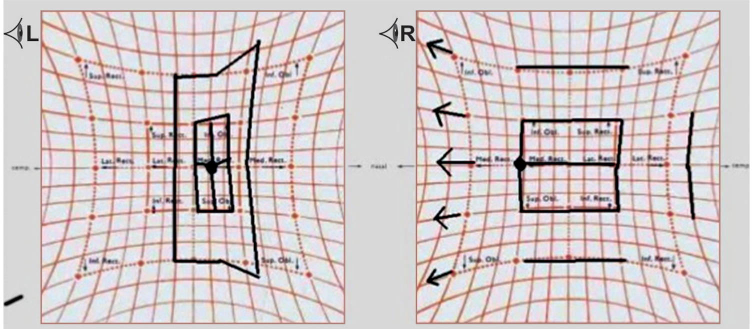
Similarities

- Both paralytic and restrictive squint present as incomitant squint (secondary deviation> primary deviation)
- Both present with diplopia in one or more gazes

Dissimilarities

But, they differ in their etiologies and clinical features

Feature	Paralytic squint	Restrictive squint
Causes	Paralysis of muscle due to damage to the nerve supplying it	Fibrosis of muscle Muscle belly caught in fracture segment
Motility vs deviation	Deviation of the eyes in the primary position corresponds to the amount of limitation movement.	Deviation of eyes in the primary position does not correspond to the amount of restriction. It is usually lesser than the amount of restriction
Forced duction test	Negative	Positive



☐ Underaction: Lt LR ☐ Overaction: Rt MR

13. Identify the test. State the diagnosis based on the result. (1+2 marks)

The Test is

It is the recording of Hess' charting.

Diagnosis

Acute right lateral rectus palsy, as the right eye has underaction of the right lateral rectus and the left eye has overaction of the medial rectus.

14. Define stereopsis. Enumerate two tests for the same. (1+2 marks)

Stereopsis

- The ability of both the eyes to perceive the depth of an object, i.e. in the third dimension
- The normal value of stereopsis is 20 seconds of an arc or better

Tests Used to Measure Stereopsis

1. Titmus fly test
2. TNO test

COMPETENCY

OP9.3 DESCRIBE THE ROLE OF REFRACTIVE ERROR CORRECTION IN A PATIENT WITH HEADACHE AND ENUMERATE THE INDICATIONS FOR REFERRAL

SHORT ESSAYS

1. Enumerate ophthalmic causes of headache. Describe the headache that is seen in uncorrected refractive errors. (3+2 marks)

Ophthalmic Causes of Headache

I. Orthoptic Causes

1. Uncorrected phorias
2. Accommodative insufficiency
3. Accommodative spasm

II. Refractive Errors

1. Presbyopia
2. Hypermetropia
3. Astigmatism
4. Myopia

III. Anterior Segment Causes

1. Corneal ulcers
2. Anterior uveitis
3. Angle-closure glaucoma
4. Juvenile open-angle glaucoma

IV. Other Causes

1. Herpes-zoster ophthalmicus
2. Migraine

Headache in Refractive Error

- Refractive error forms a major cause for headache
- When the image formed is not clear, the ciliary muscles try to bring the object into focus by contraction and relaxation
- As a result, they go into spasm at the end of the day and give rise to a headache
- Thus, the features of headache due to refractive error are headache following prolonged work to the eye and gets worse by evening

Clinical Features

- The pain is mostly described around the eyes and brow
- The pain subsides with rest or sleep
- Headache is most commonly seen with hypermetropia, followed by astigmatism and myopia

2. Enumerate four causes of primary headache. Describe the clinical features of migraines. (2+3 marks)

Causes of Primary Headache

- Primary headaches are conditions that do not have any underlying organic (ocular/neuroogical) pathology
- Causes are:
  1. Migraine
  2. Cluster headache
  3. Primary stabbing headache
  4. Trigeminal neuralgia

Clinical Features of Migraine

Symptoms

- Migraine is present in around 10% of the population and affects women more
- There is usually a strong family history and the age of onset is usually in the second-fourth decade
- Usual triggers are sleeplessness, stress, menstruation, alcohol, caffeine (sometimes), chocolate ingestion and varies with individuals
- A typical attack begins with a visual or auditory hallucination which is called the aura
- The common visual aura is the fortification spectrum where the patient sees colorful rainbow-like images which begin in the periphery like a fort and are made of zigzag lines
- The headache is usually unilateral, in the frontotemporal region and associated with nausea and vomiting

- The individual prefers to be in darkness and avoid the sound
- Mild migraine headaches can be prevented by avoiding the common triggers
- Medications can relieve the pain of an attack of migraine. Sometimes, there may be associated third nerve palsy (ophthalmoplegic migraine)

Signs

There are no signs.

SHORT ANSWERS

1. Classify migraine. (3 marks)

Common migraine or migraine without aura	Nausea, vomiting, no aura, headache
Classical migraine (headache with aura)	Aura followed by headache with or without neurological symptoms
Acephalic migraine or migraine sine migraine	Classical migraine features without headache. May have focal neurological disturbances, more common in 40–70 years of age
Ophthalmoplegic migraine	More common in the pediatric age group Migraine headache associated with paralysis of one or more extraocular muscles
Retinal migraine	Migraine with visual loss
Basilar artery migraine	Mimics vertebra-arterial basilar insufficiency with a bilateral blurring of vision, diplopia, vertigo, gait disturbances, visual hallucinations, and dysarthria

2. Describe the clinical features of cluster headache. (3 marks)

Cluster headache is a primary headache and is called so as the episodes of headache occurs in clusters.

Symptoms

- Cluster headache is typically seen in young and adult males
- The clinical features include typically one-sided, oculotemporal, and excruciating
- It is associated with restlessness
- It begins abruptly and lasts for about 10 minutes to 2 hours, and may occur in 'clusters' many times a day

Signs

- It is associated with unilateral lacrimation, rhinorrhea, and conjunctival congestion
- There is also associated ipsilateral Horner's syndrome, i.e., miosis and mild ptosis

3. A 17-year-old has an intracranial tumor in the posterior fossa. Describe the headache that is associated with raised intracranial pressure. (3 marks)

- The headache that is associated with raised intracranial pressure is localized or generalized, typically more in the mornings and associated with nausea and projectile vomiting
- The pain increases with bending of the head
- It is associated with episodes of transient obscuration of vision lasting for 20–30 seconds and associated with a change in posture

4. Explain the management of idiopathic intracranial hypertension. (3 marks)

- Management of idiopathic intracranial hypertension is a step-wise approach
- The aim is the reduction of the CSF pressure
- The first step is the removal of causative factors like weight reduction by lifestyle modification and bariatric surgery
- The next step is pharmacotherapy
  - Systemic acetazolamide up to a maximum of 1500 mg per day
  - Other drugs that can be used are diuretics like furosemide and antiepileptics like topiramate
- Surgical procedures include:
  - Serial lumbar punctures
  - Ventriculoperitoneal shunts

COMPETENCY

OP9.4 ENUMERATE, DESCRIBE AND DISCUSS THE CAUSES OF AVOIDABLE BLINDNESS AND THE NATIONAL PROGRAMS FOR CONTROL OF BLINDNESS (INCLUDING VISION 2020)

LONG ESSAY

1. A. Classify visual impairment. (2 marks)
B. State the NPCB definition of blindness in India. (1 mark)
C. Describe the key features of vision 2020. (7 marks)

A. Classification of Visual Impairment

Category of visual impairment		Level of visual activity (snellen)
Normal vision	0	6/6 to 6/18
Low vision	1	Less than 6/18 to 6/60
	2	Less than 6/60 to 3/60
Blindness	3	Less than 3/60 (FC at 3 m) to 1/60 (FC at 1 m) or visual field between 5° and 10°
	4	Less than 1/60 (FC at 1m) to light perception or visual field less than 5°
	5	No light perception

B. National Programme for Control of Blindness (NPCB), India Definition of Blindness

- Distance visual acuity <6/60 or
- Central visual field less than 20° in the better eye

C. Vision 2020

Vision 2020 was started in 2002 with the following strategic plans:

1. **Strengthening advocacy**

   National and state level

   - Creating public awareness by frequent press releases and articles in leading newspapers regarding eye care

- Involving professional organizations like All India Ophthalmic Society (AIOS), Eye Bank Association of India (EBAI), IMA and NPCB for spreading awareness

District level

- Strengthen the functioning of District Blindness Control Society (DBCS)
- Enhancing the involvement of NGOs, local communities, and societies for eyecare provision

2. **Reduction of disease burden by a specific approach**

Approach	Targets and strategies
Cataract	• To increase the cataract surgery rate to 4500 per million per year by 2005, 5000 by 2010, 5500 by 2015, and 6000 by 2020 • To improve the visual outcome of surgery to conform to standards set by WHO (i.e., 80% to have visual outcome 6/18 or >6/18 after surgery) • IOL surgery for >80% by the year 2005 and all by the year 2010. YAG capsulotomy services at all district hospitals by 2010
Childhood blindness	• Recognition of eye disorders like refractive errors, strabismus, amblyopia, and corneal diseases each time when the child is brought at the time of immunization, at school entry, and then periodically • Treatment of preventable childhood blindness like xerophthalmia, trachoma, glaucoma, ROP and refractive errors to be done at cost-effective measures • Other curable childhood blindness due to cataract, ROP, corneal opacity, and other causes to be treated by the experts at secondary and tertiary level eye care services.
Refractive errors	• Routine visual acuity assessment and follow up in schools to detect refractive error • Refraction services to be available in all primary health centres by 2010 • Availability of low-cost, good quality spectacles for children to be insured • Low vision service centres are to be established at 150 tertiary level eye care institutions • 50 such centres are to be developed by 2010, another 50 by 2015 and the final 50 by 2020
Glaucoma	• Opportunistic screening at eye care institutions should be done in all persons above the age of 35 years, especially those with diabetes mellitus, and those with a family history of glaucoma • Community-based referral by multi-purpose workers of all persons with features suggestive of glaucoma like a diminution of vision, coloured haloes, rapid change of glasses, ocular pain, and family history of glaucoma • Opportunistic screening for glaucoma at eye camps in all patients above the age of 35 years
Diabetic retinopathy	• Awareness and education regarding retinopathy by health workers • All known diabetics visiting the primary health care to be educated and examined by eye surgeon or by the ophthalmic assistant • Provision of diagnostic and therapeutic methods at tertiary level (fundus fluorescein angiography (FFA) and laser treatment) • The aim must be to bring down surgical need by medical managing DR at the secondary level
Corneal blindness	• Vitamin A prophylaxis • Awareness and hygiene regarding eye infections • Education about eye trauma and treatment • Blanket therapy for trachoma in endemic regions

3. **Human resource development**
    - Training of nurses, ophthalmic assistants, technicians, optometrists, and orthoptists
4. **Eye care infrastructure development**
    - Establishment of 20000 vision centres, 2000 service centres, 200 training centres, and 20 centres of excellence

SHORT ESSAYS

1. Enumerate four important causes for avoidable blindness. Describe the efforts taken by the government of India to eliminate blindness. (2+3 marks)

Causes for Avoidable Blindness

1. Cataract
2. Vitamin A deficiency
3. Refractive errors
4. Trachoma

Efforts Taken by the Government of India to Eliminate Cataract Related Blindness

- The National Program for the Control of Blindness (NPCB) was launched in 1976 with the following objectives
    1. To provide comprehensive health care at all three levels of health care—primary, secondary, and tertiary
    2. To decrease the incidence of blindness from 1.31% to 0.3% by the year 2000
- As the goals were not fully achieved, a 'Vision 2020' was launched to eliminate causes of avoidable blindness by the year 2020

Central level	• Director-General of Health Services at the Department of Health, Government of India • Their objectives are: 1. Procurement of goods 2. Nonrecurring grants-in-aid to NGOs 3. Information, education, and communication 4. Organizing training courses
State-level	• State ophthalmic cell has been established • Its functions are: 1. Execution of civil works for new units 2. Repairs and renovation of existing units 3. State-level training and IEC activities 4. Management of the state project cell
District level	• Establishment of District Blindness Control Societies • Plan blindness control activities throughout the district

Preventive Actions At The Primary Level

Promotive	• Nutrition education • Improved maternal and child nutrition • Health education • Face washing • Good antenatal care • Safe water • Improved–environmental sanitation

Contd.

Preventive	• Vitamin A supplementation • Measles vaccine • Perinatal care • Avoiding unscrutinised medication in pregnancy • Avoid hypoxia at birth • Examine neonate's eyes at birth • Nutrition supplementation
Curative	• Vision screening • Providing treatment for xerophthalmia • Referral for surgery • Emergency management • Treatment for trachoma • Treatment for other common eye diseases
Rehabilitative	• Provision of low vision aids • Community-based vocational rehabilitation • Counselling of the irreversibly blind • Certification of blind and provision of remuneration • Educate about concessions

2. Describe the strategies of the Government of India for eliminating corneal blindness. (5 marks)

- The important causes of corneal blindness are infectious keratitis, trachoma, ocular injuries and keratomalacia.
- Objectives regarding corneal blindness under 'Vision 2020' in India are
  1. To decrease the prevalence of preventable and curable corneal blindness
  2. To identify the infants at risk for developing corneal problems in cooperation with the RCH program

Eye Infections

Awareness programs about hygiene and ways to prevent the spread of conjunctivitis, recognizing the severity of infections of cornea and consequences.

Eye Injuries

- Health education regarding modes of eye trauma like firecracker usage, road accidents, industrial safety
- Immediate treatment and referral when necessary

Trachoma Blindness

- Blanket therapy in endemic areas
- Prompt recognition of outbreaks and treatment
- Treatment of sequelae of trachoma like trichiasis and entropion thus avoiding corneal blindness

Xerophthalmia

Prophylaxis

1. **Short-term approach: Periodic administration of oral vitamin A**

Infants <6 months not being breastfed	50,000 IU
Infants of age 6–12 months	1 lakh IU every 3–6 months
Children more than 1 year	2 lakh IU every 6 months
Lactating mothers	20,000 IU orally once at delivery to increase secretion in breast milk

- According to CSSM protocol
  - First dose (1 lakh IU): at 9 months
  - Second dose (2 lakh IU): at 18 months
  - Third dose (2 lakh IU): at 2 years of age

2. **Medium-term approach: Fortification of food with vitamin A**
   - Long-term approach: Health education to consume vitamin A rich food

COMPETENCY

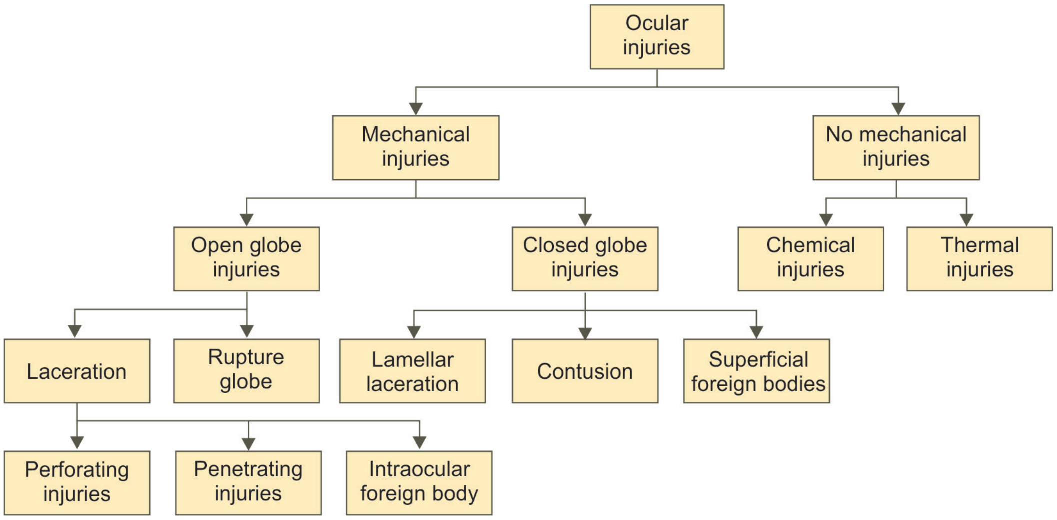
OP9.5 DESCRIBE THE EVALUATION AND ENUMERATE THE STEPS INVOLVED IN THE STABILIZATION, INITIAL MANAGEMENT AND INDICATION FOR REFERRAL IN A PATIENT WITH OCULAR INJURY

LONG ESSAYS

1. A 30-year-old man presents with injury to the right eye with a sharp piece of glass following which he has severe pain and diminution of vision.

A. Classify ocular injuries	**(3 marks)**
B. What kind of injury is this?	**(1 mark)**
C. Describe four effects of penetrating trauma on the eye.	**(4 marks)**
D. Elaborate on ocular trauma score.	**(2 marks)**
E. Explain the methods of localization of intraocular foreign bodies.	**(5 marks)**

A. Classification of Ocular Injuries Based on Bermingham Eye Trauma


Ocular injuries
Mechanical injuries
No mechanical injuries
Open globe injuries
Closed globe injuries
Chemical injuries
Thermal injuries
Laceration
Rupture globe
Lamellar laceration
Contusion
Superficial foreign bodies
Perforating injuries
Penetrating injuries
Intraocular foreign body

B. This is an Example of

Open-globe injury

C. Effects of Penetrating Trauma to the Eye

1. **Mechanical disruption**
   i. *Conjunctival laceration*
   - A partial thickness or full-thickness laceration in the conjunctiva can result from injury
   - It is associated with pricking pain in the eye and hemorrhage

- ii. *Corneal laceration*
  - Following a corneal laceration, the aqueous is drained out and thus a part of the iris plugs it and then the anterior chamber may form again
  - If it is in the pupillary area, the anterior chamber will remain flat and the eye is very soft
- iii. *Injury of sclera*
  - Usually associated with severe damage to the globe and injury to the cornea as well
  - Visual prognosis depends on the severity of the injury and involvement of the posterior segment
- iv. *Injury to the lens*
  - A breach in the lens capsule causes hydration of the cortex and swelling of the lens
  - If the injury of the lens is severe and associated with vitreous loss, the visual prognosis is guarded
- v. *Severely damaged eye with a corneoscleral tear,* with vitreous loss and uveal prolapse, has poor visual prognosis
- vi. *Intraocular foreign body*
  - It may occur following a penetrating or a perforating injury. The effects of an intraocular foreign body are mechanical disruption of anatomy, the introduction of infection, and specific reactions to metals

2. **Introduction of infection**
   - Infection of the contents of the eyeball is called endophthalmitis and inflammation of all coats of the eyeball is called panophthalmitis
3. **Posttraumatic iridocyclitis**
   - It is seen in blunt as well as penetrating trauma
   - Sometimes, it may become difficult to distinguish from endophthalmitis as both are associated with penetrating injury
4. **Sympathetic ophthalmia**
   - It is a hypersensitivity reaction that is caused by exposure of uveal proteins during a penetrating trauma.
   - It is thought to be due to a T-cell-mediated response driven against sensitized uveal antigens.
   - Viral infection may be initiating factor.
   - It is a severe, potentially blinding, bilateral granulomatous panuveitis induced by the injured eye
   - The injured eye is called the inciting eye and the other eye is called the sympathizing eye

D. Ocular Trauma Score

- It is used to prognosticate visual outcomes following eye trauma based on clinical presentation. Each parameter is given a specified raw point
- The total score is calculated, and the visual prognosis is based on the score
- The parameters are:

Initial visual acuity	No PL	60
	PL or HM	70
	1/200–19/200	80
	20/200–20/50	90
	>20/40	100

Contd.

Globe rupture	–23
Endophthalmitis	–17
Perforating injury	–14
Retinal detachment	–11
RAPD	–10

Sum	OTS score	Prognosis
0–44	1	Prognosis improves with a higher score
45–65	2	
66–80	3	
81–90	4	
92–100	5	
A score of 1 has a 1% chance of achieving a final visual acuity of >20/40 A score of 5 has a 92% chance of achieving a visual acuity >20/40		

E. Methods of Localization of the Intraocular Foreign Body

Localization of foreign bodies in the eye can be done by the following methods

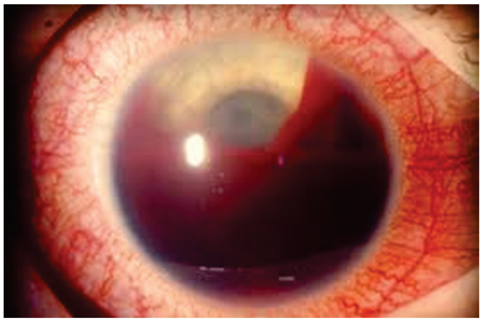
1. **X-ray**
   - Anteroposterior view and lateral views are used to detect foreign bodies
   - A limbal ring rod is used to localize the foreign body. This technique was of use before the advent of CT scans
   - It consists of a ring whose diameter is half of the diameter of the schematic eye
   - It has a perpendicular rod whose length is equal to the diameter of the ring (12 mm)
   - It is sutured at the limbus such that the ring is at the 6 o'clock position. The eye can move up and down
   - X-ray is taken with the eye looking up and the eye looking down in lateral view
   - An imaginary circle is drawn by creating a point of intersection between arcs drawn from the ends of the ring with the length of the rod as the radius
   - If the two images of the eye looking up and down are superimposed, the foreign body should be within this imaginary circle to be considered as intraocular. However, this method fails if the eyeball is too long
2. **Echography of the eyeball and the orbit**
   - This uses ultrasound waves to detect foreign bodies and helps detect and localize even non-radio-dense bodies
3. **CT scan**
   - It gives the exact location of the foreign body within the eye. However, wood cannot be detected on a CT scan
4. **MRI**
   - It is contraindicated in metallic foreign bodies. Hence, it is of last preference in detecting foreign bodies

2. A 14-year-old school boy is brought to the emergency with a history of injury to the eye with a cricket ball following which he has severe pain and swelling of lids. On examination, the anterior segment shows the following image.

A. State the diagnosis. (1 mark)

B. Explain five effects of blunt trauma on the eye. (5 marks)

C. Explain the evaluation and management of this patient? (4 marks)



A. Diagnosis

Posttraumatic hyphema

B. Effects of Blunt Trauma

Cornea	• Superficial abrasions, recurrent epithelial erosions (due to loosely attached epithelium), lamellar corneal tear, corneal blood staining can occur due to persistent hyphaema, corneal edema with Descemet's folds can be seen due to traumatic glaucoma
Anterior chamber	• Hyphaema (blood in the anterior chamber), cells, and flare can be seen in associated uveitis, depth of anterior chamber can be shallow (if associated with lens subluxation) or deep (angle recession)
Iris and pupil	• Iridodialysis (avulsion of the iris from its root) results in a D-shaped pupil • Traumatic miosis results from irritation of the sphincter due to trauma • Traumatic mydriasis—sphincter tears are visible on slit lamp as small radial tears, leading to a mydriatic pupil • Traumatic aniridia or iridemia—complete avulsion of iris • Anteflexion of iris—anterior folding of iris on itself • Retroflection of iris—posterior folding of iris on the ciliary body
Angle	• Angle recession: Cleavage between longitudinal and circular fibers
Retina	• Commotio retinae, traumatic macular hole, retinal hemorrhages, peripheral retinal tears • Retinal detachment, dialysis • Choroidal rupture: It is visible as concentric breaks in the choroid in the peripapillary area with underlying sclera visible. The visual prognosis is poor if it involves the macular area. A late complication is the formation of a choroidal neovascular membrane

C. Evaluation and Management

- The evaluation includes tonometry and dilated fundus examination
- In this case, the fundus may not be visible and a B-scan should be performed to rule out posterior segment manifestations of trauma like vitreous hemorrhage, retinal dialysis, and retinal detachment
- The patient is observed in the hospital for monitoring of IOP
- The patient is advised to bed rest in a propped-up position so that the cells settle down and vision clears
- Topical steroids and cycloplegics are given to decrease intraocular inflammation
- Anti-glaucoma medications are given to decrease intraocular pressure as hyphema is usually associated with raised IOP

- Drugs like beta-blockers, alpha agonists, carbonic anhydrase inhibitors are useful. Cholinergic and prostaglandins should not be used
- Antifibrinolytics like e-amino caproic acid and tranexamic acid can be given to prevent rebleed
- Surgical treatment is required only if IOP is very high or the hyphema does not dissolve even after 7 days. The clot is evacuated by paracentesis
- He is followed up regularly to look for associated injuries that are most commonly associated with subluxated lens and cyclodialysis

3. A 19-year-old college-going girl is brought to the emergency with a history of an acid attack by a fellow student 2 hours back. She is in severe pain and cannot open her eyelids. On examination, there are bilateral corneal clouding with total epithelial loss and limbal ischemia for 3 clock hours. However, the iris details are visible.

A. State the grade of chemical injury in this patient. (1 mark)

B. Describe the management of this condition. (4 marks)

C. Explain the legal implications for an acid attack. (3 marks)

D. Enumerate two ophthalmic conditions that constitute a grievous injury. (1 mark)

A. Grade of Chemical Injury

- The patient has corneal clouding with iris details visible
- The limbal ischemia is only 3 clock hours, so it is less than 1/3rd circumference
- It is a grade II chemical injury according to the Roper–Hall classification

B. Management

The management of this condition should be started on an emergent basis

1. **Immediate**
   - The pH of the tissue is tested with litmus paper
   - Copious wash with clean water, up to 1–1.5 L is used to wash the ocular surface
   - The flow should be continuous, hence an infusion cannula is used to irrigate all the parts of the conjunctiva, by everting the lids and irrigating the fornices
   - After 5 minutes, pH is tested again. Irrigation is continued till pH is neutralized
   - Any chunks of the chemical if found have to be removed
2. **Early**
   - Topical steroids are given every half-hourly to one hourly along with cycloplegics and antibiotics
   - Since there is an epithelial loss, a bandage contact lens is placed to improve healing
   - Forniceal sweeping twice a day with lubricants and a glass road to prevent symblepharon formation
3. **Late: After the development of complications**
   - Secondary glaucoma: Beta-blockers
   - Symblepharon: Excision of tissue
   - Limbal stem cell deficiency: It is treated with limbal stem cell graft
   - Non-healing corneal erosions: Bandage contact lens, amniotic membrane transplantation
   - Corneal opacity: Penetrating keratoplasty

C. Legal Implications of Acid Attack

- Whoever causes permanent or partial damage or deformity to, or burns or disfigures to any part/parts of the body by throwing acid to cause harm is liable for punishment
- Section 326 A in the Indian Penal code lays down the punishment of a minimum of 10 years imprisonment which can extend to life imprisonment and with fine
- The fine should be just and reasonable to meet the medical expenses of the patient
- Act 326 B lays down guidelines for an attempted acid attack

D. Grievous injury

According to section 320 of the Indian Penal code, grievous injury is defined (for the eye) as

- Permanent privation of the sight of either eye
- Permanent disfiguration of the face due to injury to the lids, orbit, or eyeball

4. A 5-year-old child is brought with a 2-day-old injury to his left eye with a metal piece. On examination, there is lid edema, congestion of the conjunctiva, and a sclerocorneal tear at 3 o'clock position with uveal prolapse. The edges of the wounds are swollen, irregular, and lined by pus. Distant direct ophthalmoscopy shows a yellow pupillary reflex.

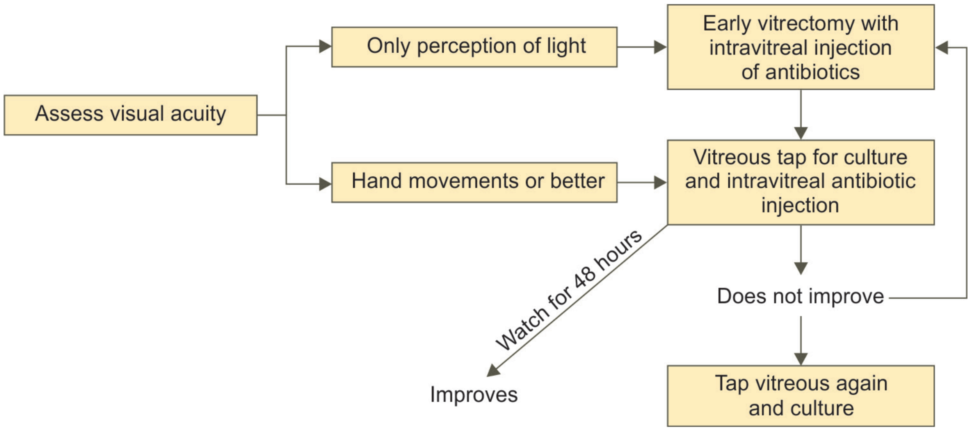
A. State the most probable diagnosis. (1 mark)
B. Explain the management of this condition? (4 marks)
C. Explain the principles of repair of sclerocorneal lacerations. (3 marks)
D. Explain the mechanism of globe rupture. (2 marks)

A. Most Probable Diagnosis

Posttraumatic endophthalmitis.

B. Management of Posttraumatic Endophthalmitis

- Posttraumatic endophthalmitis is a dreaded complication of open globe injury
- Injection tetanus toxoid and broad-spectrum systemic cephalosporins are given
- Intravitreal injection of broad-spectrum antibiotics is given in the pars plana region (3 mm behind limbus in pseudophakic eyes, 3.5 mm behind limbus in phakic eyes)
    - The usual antibiotics preferred are vancomycin (0.1 mg/0.1 ml) and ceftazidime (2.25 mg/0.1 ml)
    - Other options are vancomycin + amikacin (0.4 mg/0.1 ml) and vancomycin + gentamicin (0.2 mg/0.1 ml)
- Subconjunctival injection of a combination of antibiotics with steroids is a form of adjunct therapy


Assess visual acuity
Only perception of light
Hand movements or better
Early vitrectomy with intravitreal injection of antibiotics
Vitreous tap for culture and intravitreal antibiotic injection
Watch for 48 hours
Improves
Does not improve
Tap vitreous again and culture

  - It is given for a week to maintain intraocular concentration
  - Vancomycin 25 mg/0.5 ml + ceftazidime 100 mg/0.5 ml
  - Vancomycin + cefuroxime 125 mg/0.5 ml
  - Subconjunctival dexamethasone (4 mg/ml) is given to decrease the inflammation under the cover of antibiotics
- Topical fortified antibiotics
  - Vancomycin 50 mg/ml/or cefazoline (50 mg/ml) + amikacin (20 mg/ml)/tobramycin (15 mg%)
  - Topical steroids like prednisolone and dexamethasone are frequently instilled.
- A study—endophthalmitis vitrectomy study (EVS)—outlines the treatment protocol for postoperative endophthalmitis and the same can be followed for posttraumatic endophthalmitis

C. Principles of Repair of Sclerocorneal Lacerations

- The wound is irrigated thoroughly with saline and the necrotic tissue is excised. Iris is abscised and the ciliary body is reposited
- If any vitreous strands are seen, they are amputated
- The limbus should be first approximated using 8–0 or 9–0 silk or nylon sutures
- This is followed by the closure of the sclera using the same sutures in an anterior-to-posterior direction by mattress sutures for higher stability. This is especially important in pediatric cases
- The cornea is last to be sutured and is sutured using 9–0 or 10–0 nylon. It is closed from the limbus inwards
- The suture lengths should decrease towards the center
- After suturing, the anterior chamber is formed and leaks should be looked for
- This is followed by intravitreal injection or vitrectomy depending on the decision made

D. Mechanism of Globe Rupture

- It occurs due to the compression force
- Blunt trauma impact results in a momentary rise in the intraocular pressure which leads to an inside-out injury at the weakest part of the eyewall, i.e., in the vicinity of the canal of Schlemm concentric to the limbus (Fig. 9.5.1)

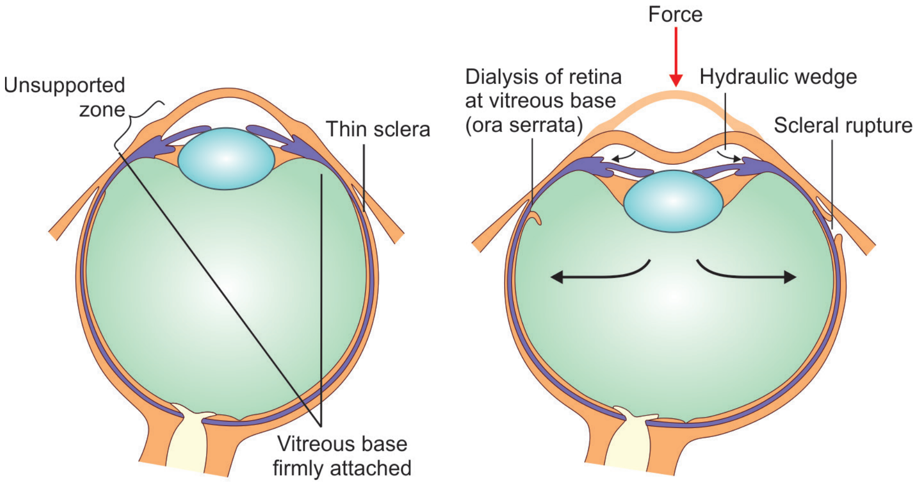

Unsupported zone
Thin sclera
Vitreous base firmly attached
Force
Dialysis of retina at vitreous base (ora serrata)
Hydraulic wedge
Scleral rupture

Fig. 9.5.1: Mechanism of globe rupture

- It occurs most commonly at the superonasal limbus as a part of the contrecoup effect—the inferotemporal quadrant being most exposed to trauma
- Rupture of the globe may be associated with prolapse of uveal tissue, vitreous loss from the wound associated with other manifestations like vitreous hemorrhage, and dislocation of the lens

SHORT ESSAYS

1. Describe the effects of blunt trauma in the eye. (5 marks)

Lids	• Ecchymosis, traumatic ptosis, periorbital edema due to accumulation of fluid as a result of trauma
Conjunctiva	• Subconjunctival hemorrhage
Cornea	• Superficial abrasions, recurrent epithelial erosions (due to loosely attached epithelium), lamellar corneal tear, corneal blood staining can occur due to persistent hyphema, corneal edema with Descemet's folds can be seen due to traumatic glaucoma
Sclera	• Partial-thickness scleral tear
Anterior chamber	• Hyphema (blood in the anterior chamber), cells, and flare can be seen in associated uveitis, depth of anterior chamber can be shallow (if associated with lens subluxation) or deep (angle recession)
Iris and pupil	• Iridodialysis (avulsion of the iris from its root) results in a D- shaped pupil • Traumatic miosis results from irritation of the sphincter due to trauma • Traumatic mydriasis—sphincter tears are visible on slit lamp as small radial tears, leading to a mydriatic pupil • Traumatic aniridia or iridemia—complete avulsion of iris • Anteflexion of iris—anterior folding of iris on itself • Retroflection of iris—posterior folding of iris on the ciliary body
Angle	• Angle recession—cleavage between longitudinal and circular fibers
Lens	• Vossius ring—an imprint of a small pupil on the lens is visible as a circular arrangement of pigments, usually smaller than the pupil as the pupil constricts during trauma • Concussion cataract—cataract formation can occur either due to a breach in the capsule or due to mechanical disturbances • A breach in the capsule leads to the imbibition of aqueous into the cortical matter leading to cataract formation and swelling • This can lead to the formation of phacomorphic glaucoma • If there is a preexisting cataract, the process will be hastened by blunt trauma • Rosette cataract is a typical feature of blunt trauma characterized by radiating opacities in the posterior subcapsular region due to the distribution of forces along the suture lines • Traumatic absorption is commonly seen in children and leads to aphakia • Subluxated lens • Dislocated lens: Total loss of zonules is called dislocation, it can be anterior or posterior. The lens can be totally in the anterior chamber or in the vitreous. They both are to be removed by intraocular surgery
Vitreous	• Liquefaction of vitreous with pigments embedded • Posterior vitreous detachment and vitreous hemorrhage

Contd.

Retina	• Commotio retinae, traumatic macular hole, retinal hemorrhages, peripheral retinal tears • Retinal detachment, dialysis • Choroidal rupture: It is visible as concentric breaks in the choroid in the peripapillary area with underlying sclera visible. The visual prognosis is poor if it involves the macular area. A late complication is the formation of a choroidal neovascular membrane
Optic nerve	• Traumatic optic neuropathy and avulsion of the optic nerve
Orbital	• Fractures, emphysema, and orbital hemorrhage

2. Describe the effects of the intraocular foreign body on ocular tissues. (5 marks)

- The intraocular foreign body may occur following a penetrating or a perforating injury
- The effects arise due to:
  1. Mechanical disruption of the anatomy
  2. Introduction of infection
  3. Reaction to the foreign body
  4. Posttraumatic iridocyclitis
  5. Sympathetic ophthalmia

1. **Mechanical disruption**
   - It depends on the size and velocity at which the foreign body enters the eye (Fig. 9.5.2)
   - The bigger the foreign body, the higher is the magnitude of the injury
   - Higher the velocity, deeper in the penetration, and higher is the damage occurring
   - The following diagram shows various pathways of entry of foreign bodies in various tissues of the eye

     A: Cornea → iris → lens → vitreous → retina, choroid, sclera
     B: Sclera → choroid → retina → vitreous
     C: Cornea → pupil → lens → vitreous
     D: Cornea → iris → zonules → vitreous

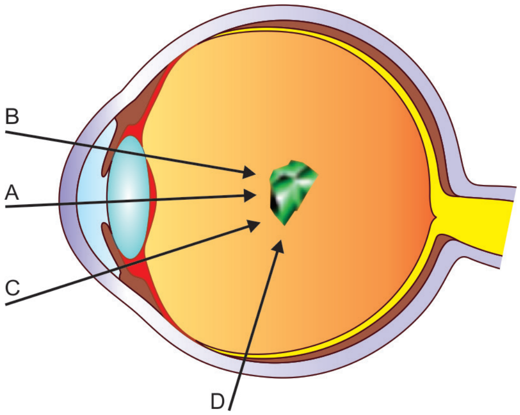


Fig. 9.5.2: Routes of entry of a foreign body

2. **Infection**
   - A metallic foreign body entering with very high velocity does not cause infection as it gets heated up en route into the eye and is thus sterilized
   - The vegetative matter may be associated with the entry of fungi
3. **Reactions to the foreign body**
   - No reaction: Glass, plastic, porcelain, gold, silver, and platinum
   - Encapsulation: Lead and aluminum
   - Sterile endophthalmitis: Pure copper, zinc, mercury, nickel
   - Specific reactions
     - Iron—siderosis bulbi
     - Copper alloys—chalcosis

4. **Posttraumatic iridocyclitis**
    - It is seen in blunt as well as penetrating trauma
    - Sometimes, it may become difficult to distinguish from endophthalmitis as both are associated with penetrating injury
5. **Sympathetic ophthalmia**
    - It is a hypersensitivity reaction that is caused by exposure of uveal proteins during a penetrating trauma
    - It is thought to be due to a T cell-mediated response driven against sensitized uveal antigens
    - Viral infection may be initiating factor. It is a severe, potentially blinding, bilateral granulomatous panuveitis induced by the injured eye
    - The injured eye is called the inciting eye and the other eye is called sympathizing eye

3. Describe the clinical features of siderosis bulbi. (5 marks)

Siderosis Bulbi

- It is a very specific reaction of the ocular tissue to iron and iron-containing alloys
- It occurs due to the dissociation of ferrous ions into the ocular tissues and combines with intracellular proteins leading to their degeneration
- It occurs around 2 months following the introduction of the foreign body

Clinical Features

- Cornea: Brownish discoloration of the epithelium
- Iris: Reverse heterochromia—increase in pigmentation of iris
- Pupil: Mid dilated due to damage to the sphincter, failure of accommodation (earliest symptom)
- Lens: The anterior capsule of the lens shows rust-like opacities. It is the first sign of siderosis. Later, it may progress to a full-blown cataract, usually yellow with brown patches
- Chronic open-angle glaucoma: Unknown pathophysiology
- Retina: Pseudoretinitis pigmentosa like a picture, due to degeneration of rods, followed by cones. Hence, the pigmentary retinopathy starts in the periphery (in contrast to RP, where it starts in midperiphery, spares macula) and progresses to affect the macula. ERG is very sensitive than visual acuity testing in picking up retinal degeneration. A reduced b wave amplitude is seen
- When these changes appear, the foreign body may not be found on imaging at all due to complete dissolution
- These changes are irreversible

4. Describe the initial evaluation and management of an eye with penetrating eye trauma. (5 marks)

Evaluation

- Patients with penetrating eye injury usually present with sudden onset pain, blurred vision, and sometimes bleed from the eye
- An eye with a full-thickness corneal tear may also give a history of a gush of fluid from the eye
- An initial assessment should include a complete history of the mode of injury, the time at which the injury occurred, and ruling out medicolegal implications if any

- Also, a history of alcohol intake, systemic illnesses must be noted as they may be important during anesthesia as most of the eye injuries are repaired under sedation or general anesthesia
- A general examination is done to rule out more life-threatening injuries
- Ophthalmic assessment includes a careful recording of visual acuity in the injured as well as the healthy eye. Slit-lamp examination is mandatory to understand the depth, site, and severity of the wound
- The presence of any uveal prolapse must be noted
- At no point, intraocular pressure must be assessed as the contents of the eye can be forced out during the procedure
- A transillumination test should be performed using retroillumination in the slit lamp. This is especially important if the history is suggestive of the entry of a foreign body
- Fundus examination in the present state is done to see the health of the disc
- A CT scan is performed to rule out the presence of an intraocular foreign body

Management

- Depending on the severity and nature of the injury, the patients are counseled regarding the repair of the wound
- It is preferably done within 6 hours of injury

Clinical scenario	Management principles
Corneal tear, <2 mm, anterior chamber well-formed, clean wound, no foreign body	Instill sterile antibiotics and cycloplegics Pressure bandage of the eye, wait for 24 hours
Corneal tear, >2 mm, anterior chamber well-formed, clean wound, no foreign body	Consider repair under local/general anesthesia
Corneal tear, >2 mm, anterior chamber collapsed, no uveal prolapse, clean wound, no foreign body	Emergency repair, preferably under general anesthesia A corneal laceration is repaired with 10–0 nylon sutures and an anterior chamber is formed
Corneal tear, >2 mm, anterior chamber collapsed with iris prolapse, clean wound, no foreign body	Emergency repair, preferably under general anesthesia Corneal laceration after iris abscission, repaired with 10–0 nylon sutures, and the anterior chamber is formed
Corneal tear, any size, regardless of the status of the anterior chamber, with foreign body	Emergency repair, preferably under general anesthesia Removal of a foreign body by paracentesis, corneal laceration is repaired with 10–0 nylon sutures and the anterior chamber is formed
Corneal tear, >2 mm, with traumatic cataract, anterior chamber deep	Emergency repair, preferably under general anesthesia Lens aspiration followed by corneal laceration is repaired with 10–0 nylon sutures and the anterior chamber is formed. IOL placement planned later
Corneal tear, with intravitreal foreign body	Emergency repair, preferably under general anesthesia Corneal tear repair with vitrectomy

5. Describe the initial evaluation and management of an eye with blunt eye trauma. (5 marks)

Evaluation

- Patients with blunt eye injury usually present with sudden onset pain, blurred vision, and may be associated with a head injury as well
- After ruling out head injury and other major systemic injuries, an ophthalmic assessment should proceed
- Medicolegal issues must be given due detailing
- An initial assessment should include a complete history of the mode of injury, time at which the injury occurred
- Ophthalmic assessment includes a careful recording of visual acuity in the injured as well as the healthy eye
- Slit-lamp examination is mandatory to understand various aspects of trauma
- Fluorescein staining helps to identify any minute corneal abrasions or even sites of globe rupture
- Pupil reactions are very important, and a swinging flashlight test should be performed
- Intraocular pressure and fundus examination must be carried out meticulously
- The bony margins of the orbit should be palpated to look for discontinuity or crepitus
- Tenderness may be present due to trauma but does not necessarily suggest a fracture
- B-scan of the eyeball and the orbit are done in cases of doubt. CT scan helps to rule out orbital fractures

Management

Globe rupture	Analgesics, antiemetics. Repair of the ruptured segment as a corneoscleral tear
Retrobulbar hematoma	Analgesics, antiemetics, lateral canthotomy, intravenous mannitol
Hyphema	Bed rest in propped up position, antiglaucoma medications, topical steroids, and cycloplegics
Subluxation of lens	Mild degrees: Correction of astigmatism Moderate degrees: Lens extraction with IOL Severe degrees: ICCE + vitrectomy + scleral fixated IOL
Retinal dialysis	LASER retinopexy + scleral buckling
Vitreous hemorrhage without retinal detachment	Bed rest in propped up position Consider vitrectomy if hemorrhage does not recede in 3 months
Vitreous hemorrhage with retinal detachment	Pars plana vitrectomy with scleral buckle
Traumatic optic neuropathy	Trial of intravenous steroids Orbital canal decompression

6. Describe the initial evaluation and management of an eye with the chemical injury. (5 marks)

- Evaluation of an eye with chemical injury should follow immediate copious irrigation with normal saline and removal of any flecks of chemical from the ocular surface
- One must not waste valuable time doing a thorough examination before irrigation
- The nature of the chemical that has caused the injury must be enquired about

Immediate Management

- The pH of the tissue is tested with litmus paper. Copious wash with balanced salt soultion up to 1–1.5 L is used to wash the ocular surface
- The flow has to continuous, hence an infusion cannula is used to irrigate all the parts of the conjunctiva, by everting the lids and irrigating the fornices
- After 5 minutes, pH is tested again. Irrigation is continued till pH is neutralized
- Any chunks of chemicals if found should be removed
- Some even advocate the addition of a weaker acid in case of alkali burn and vice versa to neutralize the pH
- Necrotic tissue should be excised

Evaluation

- Many a time, a chemical injury may be a medicolegal issue and careful documentation may be necessary
- The evaluation includes visual acuity assessment, slit-lamp examination, and fluorescein staining of the cornea
- Raw areas may show up as green areas in the conjunctiva and on the cornea
- The degree of limbal whitening is also noted
- Then the chemical injury is graded based on Dua’s classification

Grade	Prognosis	Clinical findings	Conjunctival involvement
I	Very good	0 clock hours of limbal involvement	0%
II	Good	≤3 clock hours of limbal involvement	≤30%
III	Good	3–6 clock hours of limbal involvement	30–50%
IV	Good–guarded	6–9 clock hours of limbal involvement	50–75%
V	Guarded–poor	9– <12 clock hours of limbal involvement	75– <100%
VI	Very poor	Total (12 clock hours) limbal involvement	100%

Early Management

- Topical steroids are given every half-hourly to one hourly along with cycloplegics and antibiotics
- Forniceal sweeping twice a day with lubricants and a glass road to prevent symblepharon formation
- Immunosuppressants can also be used in very severe cases of limbal ischemia

7. Explain the pathogenesis of chemical eye injury. (5 marks)

Pathogenesis of Chemical Eye Injury

The pathogenesis of chemical eye injury can be staged as follows:

1. **Stage of acute ischaemic necrosis**
    - Due to exposure to the caustic agent, there is a reaction between the ocular tissues and the agent
    - There is damage to the epithelium causing necrosis and occlusion of the limbal vessels
    - Deeper penetration of the agent causes stromal opacification
    - It is characterized by:
        - Conjunctival congestion, chemosis, and mucopurulent discharge, corneal epithelial sloughing, and opacification

  - Acute iridocyclitis is also seen (due to penetration into the anterior chamber)
  - Limbal whitening is seen due to ischemia of the limbus
2. **Stage of repair**
   - In this stage conjunctival and corneal epithelium regenerates, there occurs corneal vascularization, and inflammation of the iris subsides
   - However, due to loss of limbal stem cells repair is very slow and this stage may be marked by delayed epithelial healing and recurrent erosions of the corneal and conjunctival epithelium
   - There are raw surfaces in the conjunctiva that heal with fibrosis and adhesions
3. **Stage of complications**
   - As a result of the above process, symblepharon and subconjunctival fibrosis develop
   - Therefore, these patients develop entropion, ectropion, trichiasis, and recurrent corneal ulceration
   - Other complications are the development of complicated cataracts and secondary open-angle glaucoma due to shrinkage of the sclera
   - Due to the daage of the ciliary processes, hypotony and phthisis bulbi may develop

8. Explain the seven rings of campbell due to ocular trauma. (5 marks)

- 7 rings of campbell are seen in association with blunt ocular trauma
- They are classically anterior to the equator

1. **Central iris: Sphincter tear**
   - They are radial tears in the pupil
   - They do not interfere with visual function and are just a sign of blunt trauma
2. **Peripheral iris: Iridodialysis**
   - It is detachment of the root of the iris from its attachment to the ciliary body
   - It is associated with hyphema frequently as the major arterial circle is present at the root of the iris
3. **Anterior ciliary body: Angle recession**
   - It is a tear between the circular and longitudinal muscles of the ciliary body causing the widening of the ciliary body band on the gonioscopy
   - It predisposes to angle recession glaucoma
4. **Separation of the ciliary body from the scleral spur: Cyclodialysis**
   - It is a separation of the ciliary body from its attachment and is associated with low IOP
5. **Trabecular meshwork**
   - Tear in the trabecular meshwork
6. **Zonules/lens: Zonular tears with possible lens subluxation**
   - Causes subluxation or dislocation of the crystalline lens
7. **Separation of the retina from the ora serrata: Retinal dialysis**
   - Disinsertion of the retina at the ora serrata

9. Explain the effects of blunt trauma on the lens. (5 marks)

The features of blunt trauma on the crystalline lens are:

1. **Vossius ring**
   - An imprint of the small pupil on the lens is visible as a circular arrangement of pigments, usually smaller than the pupil as the pupil constricts during trauma
2. **Concussion cataract**
   - Cataract formation can occur either due to the breach in the capsule or due to mechanical disturbances

- A breach in the capsule leads to the imbibition of aqueous into the cortical matter leading to cataract formation and swelling
- This can lead to the formation of phacomorphic glaucoma
- If there is a preexisting cataract, the process will be hastened by blunt trauma
- Rosette cataract is a typical feature of blunt trauma characterized by radiating opacities in the posterior subcapsular region due to the distribution of forces along the suture lines

3. **Traumatic absorption**
   - Commonly seen in children and leads to aphakia
4. **Subluxated lens**
   - The signs of partial loss of zonules are:
     - Phacodonesis: Tremulousness of lens
     - Iridodonesis: Tremulousness of iris
     - Irregular myopic astigmatism: Even if the position of the lens does not change in mild subluxation, there is induced myopia due to an increase in anterior curvature of the lens due to the absence of tug by the zonular fibers
     - Irregular anterior chamber. It can occlude angle in one or more quadrants leading to angle closure and peripheral anterior synechiae formation
     - On dilatation, the edge of the subluxated lens is seen. Gonioscopy performed after dilatation helps in understanding the amount of zonular dialysis. The presence of vitreous herniation confirms zonular loss
     - Vitreous herniation into the anterior chamber is seen a gel-like structure entering from the pupil, may be laden with pigments, and pupil block can be caused sometimes
     - Vitreous touch syndrome: When vitreous meets the endothelium of the cornea
5. **Dislocation of the lens**
   - Total loss of zonules is called dislocation, it can be anterior or posterior
   - The lens can be totally in the anterior chamber or in the vitreous
   - They both are to be removed by intraocular surgery

10. Describe the posterior segment manifestations of blunt trauma. (5 marks)

Posterior Segment Manifestations of Blunt Trauma

Vitreous	• Liquefaction of vitreous with pigments embedded • Posterior vitreous detachment and vitreous hemorrhage
Retina/choroid	• Commotio retinae: It is the most common presentation of blunt trauma in the posterior segment. It is a relatively benign condition characterized by edema of the outer layers of the retina that subsides with time • Traumatic macular hole, retinal hemorrhages, peripheral retinal tears can also be caused • Retinal detachment • Retinal dialysis: It is a separation of the retina from its attachment at the ora serrata It is usually seen in the superonasal quadrant as trauma is more frequent in the inferotemporal quadrant • Choroidal rupture: It is visible as concentric breaks in the choroid in the peripapillary area with underlying sclera visible. Visual prognosis is poor if it involves the macular area. The late complication is the formation of a choroidal neovascular membrane

Contd.

Optic nerve	• Traumatic optic neuropathy is seen in both penetrating and blunt trauma. In blunt trauma, it is due to the transmission of forces along the cranial bones to the optic nerve. It usually carries a poor prognosis • Avulsion of the optic nerve is of rare occurrence. The optic nerve head is detached from its insertion into the globe

11. Describe the clinical features and management of subluxation of lens. (3+2 marks)

Subluxation of Lens

Clinical Features

- Subluxation of the lens is a common entity following blunt trauma
- The signs of partial loss of zonules are:
  - Phacodonesis: Tremulousness of lens
  - Iridodonesis: Tremulousness of iris
  - Irregular myopic astigmatism: Even if the position of the lens does not change in mild subluxation, there is induced myopia due to an increase in anterior curvature of the lens due to the absence of tug by the zonular fibers
  - Irregular anterior chamber. It can occlude angle in one or more quadrants leading to angle closure and peripheral anterior synechiae formation
  - On dilatation, the edge of the subluxated lens is seen. Gonioscopy performed after dilatation helps in understanding the amount of zonular dialysis. The presence of vitreous herniation confirms zonular loss
  - Vitreous herniation into the anterior chamber is seen a gel-like structure entering from the pupil, may be laden with pigments, and pupil block can be caused sometimes
  - Vitreous touch syndrome: When vitreous comes in contact

Management

- Depends on the amount of subluxation
- Minimal degrees of zonular dialysis with no complications
  - Can be left alone by refractive correction with concave cylinders
  - Miotic therapy is useful in avoiding diplopia due to the edge of the lens in the pupillary area
- Aphakic correction can be given if the aphakic area in the pupil is more significant
- Surgery (lens extraction with IOL) is indicated in
  - Pupillary block or dislocation of the lens into the anterior chamber
  - Myopia, astigmatism or monocular diplopia cannot be corrected by refraction or medical measures
  - Vitreous touching the cornea
  - The lens in the posterior segment
  - Lens or cataract extraction is done with the placement of capsular tension segments (up to 90° of zonular loss) or rings (>90°). When the capsular bag is severely compromised, ICCE with vitrectomy + scleral fixated or iris-claw lens or anterior chamber IOL is preferred

12. Describe the principles of management of corneal lacerations. (5 marks)

Principles of Management of Corneal Lacerations

- It is the most commonly seen injury in an open globe injury

- Principles of treatment are:
    1. Always rule out an intraocular foreign body by a thorough history, examination, and imaging if needed. Iris should be carefully examined to look for the entry of a foreign body. Transillumination is the best way to look for the entry points of a foreign body
    2. Patient education: Explain to the patient and bystanders, the risks of the condition that is remote chances of endophthalmitis and retinal detachment. Also, explain the probability of formation of cataracts and a need for the second surgery. The prognosis of vision may be guarded based on the location and extent of the tear
    3. Repair of the wound has to be done on an urgent basis (<6 hours)
    4. <2 mm size of corneal tears, if noninfected, need not be sutured. The eye should be padded with antibiotic drops and cycloplegics. Usually, they are self-sealing
    5. A clean noninfected corneal tear is sutured right away using 10-0 Nylon sutures, which should be removed after 6 weeks
    6. If there is an iris prolapse, it is abscised. Introduction of iris back into the anterior chamber carries a very high risk of endophthalmitis
    7. Suturing is first done to maintain continuity at the limbus and then carried on towards the center
    8. Sutures should be longer towards the periphery and shorter towards the center
        - Long sutures = good stability of the wound = high astigmatism
        - Short sutures= less stability = low astigmatism.
    9. Postoperatively antibiotic eye drops, steroid eye drops, and cycloplegics are prescribed
    10. If there is associated lens injury and leaking of cortical matter, lens removal has to be done simultaneously. IOL implantation can be planned later once the eye stabilizes so that a proper “k” reading can be taken
    11. If there is associated posterior segment involvement, corneal laceration is sutured first and then vitrectomy is performed if needed
    12. If surgery has to be postponed for some reason, always patch the eye without applying pressure.

13. Describe the clinical features and management of sympathetic ophthalmia.

(5 marks)

Sympathetic Ophthalmia

Sympathetic ophthalmia is a bilateral serious, chronic granulomatous, panuveitis due to sensitization to choroidal antigen due to either injury or surgery in the fellow eye.

Clinical Features

Prodromal stage

- When the sympathetic ophthalmia begins, there is a return of ciliary congestion, tenderness, and lacrimation in the shrunken eye
- In the sympathizing eye, the first symptom to begin is photophobia and defective near vision as a result of the weakness of accommodation
- A careful examination may reveal ciliary tenderness, KPs, vitreous cells, and flare

Established stage

- Full-fledged granulomatous uveitis features may be seen: iridocyclitis, peripheral retinal exudation, Dalen Fuchs nodules, and papillitis

- It tends to be chronic and may last many months
- Inflammation may be severe enough to cause complications like phthisis

Management

Prophylaxis

- Steroid administration in every case of penetrating trauma, especially if it involves the ciliary body
- Meticulous repair of any penetrating injury to take care of the uveal tissue not to be incarcerated in the wound
- Uncontrolled uveitis in the severely damaged eye for more than 2 weeks should be enucleated

Treatment

- Early recognition of signs of inflammation and starting steroid therapy in the form of intravenous methylprednisolone 1000 mg followed by oral Prednisolone 1mg/kg and topical steroids and cycloplegics
- After the eye quietens down, a maintenance dose of 15–20 mg is administered for many months and the eye has to be monitored
- Enucleation of the exciting blind eye is proposed by some but must be done before the initiation of inflammation

14. Explain Roper–Hall and Dua's classification of chemical injury. Explain why the latter staging is better. (4+1 marks)

Roper–Hall Classification

Grade	Clinical findings		Prognosis
	Cornea	*Conjunctiva/limbus*	
I	Cornea epithelial damage	No limbal ischemia	Good
II	Corneal haze, iris details visible	<1/3 limbal ischemia	Good
III	Total epithelial loss, stromal haze, and iris details obscured	1/3–1/2 limbal ischemia	Guarded
IV	Cornea opaque, iris and pupil obscured	>1/2 limbal ischemia	Poor

Dua's Classification

Grade	Prognosis	Clinical findings	Conjunctival involvement
I	Very Good	0 clock hours of limbal involvement	0%
II	Good	≤3 clock hours of limbal involvement	≤30%
III	Good	3–6 clock hours of limbal involvement	30–50%
IV	Good–guarded	6–9 clock hours of limbal involvement	50–75%
V	Guarded–poor	9 – <12 clock hours of limbal involvement	75– <100%
VI	Very poor	Total (12 clock hours) limbal involvement	100%

Dua's Classification is Better than Roper–Hall

As it takes into account bulbar conjunctival involvement as well.

15. Enumerate the various manifestations of trauma to the eyelids. Elaborate on the repair of a full-thickness tear of the upper lid. (2+3 marks)

Manifestations of Trauma to the Eyelids

Blunt injury to the eyelids	Sharp injury to the eyelids
• Abrasions	• Lid laceration
• Edema of the lids	• Partial-thickness
• Ecchymosis of lids	• Full-thickness
• Eyelid hematoma	• With the loss of tissue
• Traumatic ptosis	• Associated lacrimal system injury

Repair of a Full-thickness Upper Lid Laceration

- A quick initial assessment of the injury is made to rule out other ocular injuries and head injuries
- Proparacaine is instilled in the conjunctival sac and the lid is anesthetized by local infiltration with a 1:1 mixture of lignocaine 2% with 1:200000 epinephrine and 0.75% bupivacaine is best suited for small lacerations
- The anesthesia is injected using a 26 gauge or 30 gauge ½ inch needle
- Suture materials
    - Nonabsorbable: Nylon, silk used for skin
    - Absorbable: Vicryl is used for subcutaneous tissue, muscle, and conjunctiva. It is also preferred in pediatric cases as suture removal might be difficult. The size preferred is 6–0 or 7–0
    - Cutting and spatulated needles are preferred

Procedure

- A lid guard is placed to protect the cornea. The lid margins are approximated first with a tight secure knot
- The lid is then sutured in layers with buried sutures for the conjunctiva-muscle layer and then followed by nonabsorbable interrupted sutures for the skin (Fig. 9.5.3)
- The area is padded with antibiotic ointment and analgesics are given. Suture removal is done on the eighth day

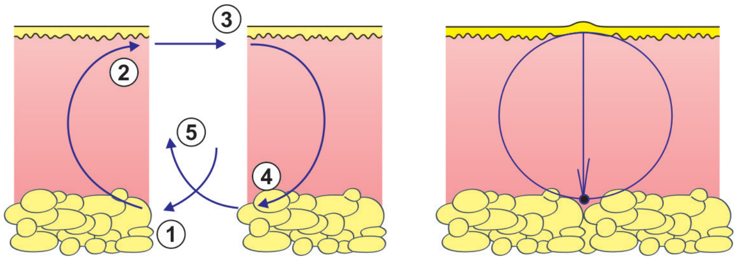


Fig. 9.5.3: Method of suturing a full-thickness lid laceration

16. Describe the etiology, clinical features, and management of traumatic optic nerve injuries. (1+2+2 marks)

Traumatic Optic Nerve Injuries

Etiology

- An important cause of permanent visual loss as around 50% of patients have visual loss as low as the perception of light only
- Can occur due to a blow to the frontal area of the head, severe enough to cause loss of consciousness.
- The shearing forces of the movement of the brain cause movement of the intracanalicular portion of the optic nerve (the most common site of injury in indirect trauma) or by direct transection of the optic nerve (rare)
- In indirect trauma, there is a shearing of pial vessels which leads to loss of blood supply and thus optic neuropathy

Clinical Features

- Clinical features are similar to all optic neuropathies: Decreased vision (varies with the amount of nerve fibers damaged), RAPD, visual field defect (depends on the amount of loss of nerve fibers), dyschromatopsia
- When the optic disc is avulsed from the eyeball, it is seen as a ring of hemorrhage around the optic disc area
- If the site of injury is distal to the entry of CRA, then it may appear as a central retinal artery occlusion
- The optic disc appears normal (at the time of injury) when the site of injury is proximal to the entry of the central retinal artery-most common. Later, optic atrophy develops

Management

- Three days of intravenous methylprednisolone 1g QID followed by 13 days of oral prednisolone 1 mg/kg followed by a short taper has been tried with some success
- The National Acute Spinal Cord Injury Study II (NASCIS) concluded the use of high dose steroids and found an improvement in motor and sensory aspects following spinal cord injury and thus has been used for traumatic optic neuropathy as well
- Optic nerve decompression is indicated through a transethmoidal route or transfrontal route

17. A 45-year-old man presented to the emergency with injury to the right eye during lighting a firecracker in an inebriated state. Ocular examination shows a 5 mm long corneoscleral tear at 5o'clock associated with uveal prolapse. Explain the management of this patient. (5 marks)

Assessment

- An initial assessment should include a complete history of the mode of injury, the time at which the injury occurred, and ruling out medicolegal implications if any
- Also, a history of alcohol intake, systemic illnesses must be noted as they may be important during anesthesia as most of the eye injuries are repaired under sedation or general anesthesia
- Ophthalmic assessment includes a careful recording of visual acuity in the injured as well as the healthy eye. Slit-lamp examination is mandatory to understand the depth, site, and severity of the wound

- The presence of any uveal prolapse must be noted
- The repair is performed in a sterile environment with the help of an operating microscope
  - The wound is irrigated thoroughly with saline and the necrotic tissue is excised. Iris is abscised and the ciliary body is reposited. If any vitreous strands are seen, they are amputated
  - The limbus should be first approximated using 8–0 or 9–0 silk or nylon sutures
  - This is followed by the closure of the sclera using the same sutures in an anterior-to-posterior direction by mattress sutures for higher stability
  - The cornea is last to be sutured and is sutured using 9–0 or 10–0 nylon
  - It is closed from the limbus inwards
  - The suture lengths should decrease towards the center
  - After suturing, the anterior chamber is formed and leaks should be looked for

18. Categorise the sites of lodgment of extraocular foreign bodies. Describe their clinical features. (2+3 marks)

Sites of Lodgment of Extraocular Foreign Bodies

- An extraocular foreign body can get lodged in the conjunctiva or cornea
- Possible sites in the conjunctiva
  1. Fornix
  2. Sulcus subtarsalis
  3. Subconjunctival space following a conjunctival laceration
  4. Bulbar conjunctiva
- Possible sites in the cornea
  1. Epithelium
  2. Stroma
  3. Limbus
- Commonly encountered foreign bodies are dirt, iron particles (in industrial workers), husk of paddy, wings of insects, and sand
- They usually incite irritation and rarely can even lead to an allergic response (caterpillar hair)

Clinical Features

- Patients present with sudden onset of irritation, lacrimation, photophobia, and redness in the eye
- Corneal foreign bodies are extremely painful as the cornea has the densest innervation in the body
- If the foreign body is in the pupillary area, it can cause decreased vision
- The examination is done thoroughly to look for the foreign body
- Instilling fluorescein helps to localize the foreign body based on the pattern of corneal abrasions
- Complications
  - They have a propensity to cause infection (conjunctivitis, keratitis)
  - Corneal foreign bodies can lead to the formation of an ulcer or an opacity which, if in the pupillary area, can lead to the diminution of vision
  - In iron foreign bodies, a rust ring (brownish discoloration) forms around the foreign body due to the diffusion of ferrous ions into the stroma

SHORT ANSWERS

1. Contrast central retinal artery occlusion (CRAO) and commotio retinae. (3 marks)

Both early CRAO and commotio retinae have a cherry-red spot. Hence their differentiation is necessary

Feature	CRAO	Commotio retinae
History	Sudden painless loss of vision	History of trauma followed by diminished vision
Vision	Very low	Variable, may be normal or as low as 6/60
Pupil	RAPD	No RAPD
Arteries	Narrowed Cattle-trucking Emboli may be seen	Normal No emboli are seen in arteries
Visual prognosis	Poor	Very good
FFA	Delayed arm-retina circulation time Poor filling of arteries	Normal arm-retina circulation time Normal filling of arteries
ERG	Poor response	Normal

2. Describe the features of chalcosis bulbi. (3 marks)

- Chalcosis is a specific reaction of the ocular tissues to copper alloys. Pure copper does not cause chacosis
- It occurs due to the deposition of electrolytically dissociated copper ions in various membranes of the eye
- There is no intracellular interaction and associated degeneration as in siderosis
  1. *Cornea:* Copper is deposited in the Descemet's membrane causing a golden brown Kayser-Fleischer ring
  2. *Lens:* Copper deposition underneath the posterior capsule of the lens leads to sunflower cataract formation
  3. *Retina:* Golden plaques at the posterior pole with a metallic sheen

3. Describe the measures to be taken to prevent sympathetic ophthalmia in a case of open globe injury. (3 marks)

- The incidence of sympathetic ophthalmia is higher when there is an injury in the ciliary zone and more so when there is a uveal prolapse
- Steroid administration in every case of penetrating trauma, especially if it involves the ciliary body. Steroids are given as topical and systemic preparation
- Meticulous repair of any penetrating injury to take care of the uveal tissue not to be incarcerated in the wound
- Uncontrolled uveitis in the severely damaged eye for more than 2 weeks should be enucleated

4. Alkali injury is worse than acid burns. Justify this statement. (3 marks)

- Alkalis combine with fatty acids of the cell membrane and lead to saponification and destruction of the structure of the cell membrane. The tissues as a result get softened and gelatinized
- They are hygroscopic and therefore they dehydrate the cells and lead to necrosis of ocular tissue
- Also, there is deeper penetration of the alkalis into the tissues (cornel stroma, anterior chamber, and ciliary body)

- Damage is widespread and continues even after the offending agent is removed
- Acids react with the cells and cause instant coagulation which forms a mechanical barrier for further penetration
- Acid injuries are less notorious than alkalis as the inflammation is not as widespread as that due to alkalis

5. Describe the clinical features of commotio retinae. (3 marks)

- Commotio retinae is also called Berlin's edema and one of the most common manifestations of blunt trauma
- It is characterized by edema of the outer layers of the retina involving varying amounts of the posterior pole
- It is visible as a geographic area of whitening of the retina with a "pseudo-cherry-red spot" (excepting the fovea which appears red) with ill defines margins
- The cause for this is, rebound mechanical shock waves that damage the outer layers of the retina, i.e., RPE-photoreceptor complex
- Visual acuity is variable and depends on the amount of edema
- The prognosis is excellent as the edema resolves over a few days
- A few patients may develop pigmentary changes in the retina and may be associated with a diminution of vision

6. Explain the mechanism of closed globe injury. (3 marks)

Mechanism of Closed Globe Injury in Blunt Trauma (Fig. 9.5.4)

- The mechanism of closed globe injury can be explained in the following steps
- Direct compressive forces over the outer wall
- Maximum damage is borne by the site of injury
- Distribution of the force to various tissues
  - The force is transmitted through the ocular fluids to angle, iris-lens diaphragm, retina, and choroid
- Reflection of the shock waves to posterior pole
  - The force is transmitted to the posterior pole, mainly converging on to the macula/posterior pole
- Rebound of vector forces back to the anterior segment
  - When the force travels anteriorly again, it pulls on the retina-choroid complex, iris-lens diaphragm

7. Enumerate three inert intraocular foreign bodies. (3 marks)

1. Gold
2. Silver
3. Plastic

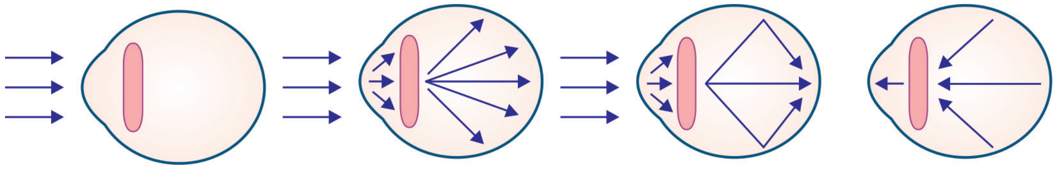


Fig. 9.5.4: Mechanism of closed globe injury

8. Contrast between siderosis and chalcosis bulbi. (3 marks)

Feature	Siderosis bulbi	Chalcosis
Cause	Iron alloy	Copper alloy
Mechanism of damage	• It occurs due to the dissociation of ferrous ions into the ocular tissues that get converted into ferric ions that combine with intracellular proteins to release hydroxyl ions leading to tissue degeneration.	• It occurs due to the deposition of electrolytically dissociated copper ions in various membranes of the eye.
Clinical features	• Cornea: Brownish discoloration of the epithelium • Iris: Reverse heterochromia-increase in pigmentation of iris. • Pupil: Mid dilated due to damage to the sphincter, failure of accommodation (earliest symptom) • Lens: The anterior capsule of the lens shows rust-like opacities. It is the first sign of siderosis. Later, it may progress to full-blown cataracts, usually yellow with brown patches. • Chronic open-angle glaucoma: unknown pathophysiology. • Retina: Pseudoretinitis pigmentosa like picture	• Cornea: Copper is deposited in the Descemet membrane causing a golden brown Kayser–Fleischer ring. • Lens: Copper deposition underneath the posterior capsule of the lens leads to sunflower cataract formation • Retina: Golden plaques at the posterior pole with a metallic sheen.
Causes irreversible visual loss	Yes	No
Treatment	• Remove FB if seen on imaging • Galvanic deactivation • Administration of intravenous EDTA • Desferrioxamine	• Remove FB if seen on imaging • Sodium thiosulphate • BAL (British Anti-Lewisite)

9. Describe the long-term effects of a caustic eye injury. (3 marks)

- Following a chemical injury and the resultant inflammation, there is a loss of the limbal stem cells and the repair process is very slow
- There are raw surfaces in the conjunctiva that heal with fibrosis and adhesions
- As a result of the above process, symblepharon and subconjunctival fibrosis develop
- Therefore, these patients develop entropion, ectropion, trichiasis, and recurrent corneal ulceration
- Other complications are the development of complicated cataracts and secondary open-angle glaucoma due to shrinkage of the sclera
- Due to corneal opacification, there is a great risk of visual loss.
- Keratoplasty has high risks due to the loss of limbal stem cells. Keratoprosthesis is a better option in these patients

10. Describe the changes in the pupil following blunt trauma. (3 marks)

- Immediately following blunt trauma, the pupil constricts (miosis) and leaves an imprint on the anterior capsule of the lens
- This is followed by mydriasis. This may be permanent and is called traumatic cycloplegia. Unilateral mydriasis may also be a sign of head injury

- Sphincter tears cause radial cuts in the pupil
- A D-shaped pupil is seen in iridodialysis. The flat of the 'D' suggests the corresponding site of dialysis
- In severe blunt trauma and associated globe rupture with iris prolapse, the pupil is peaked to the direction of the prolapse (Fig. 9.5.5)

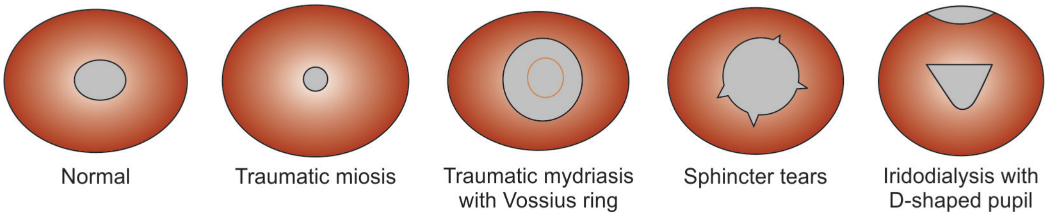

Normal
Traumatic miosis
Traumatic mydriasis with Vossius ring
Sphincter tears
Iridodialysis with D-shaped pupil

Fig. 9.5.5: Changes in the pupil after blunt trauma

11. Explain the mechanism and clinical features of angle recession glaucoma. (1+2=3 marks)

Angle Recession Glaucoma

Mechanism

- Angle recession occurs due to blunt trauma leading to the formation of cleavage between longitudinal and circular muscles leading to deepening of the anterior chamber
- With time (may take years together), scarring takes place leading to a decrease in filtering capacity
- 6–9% of individuals with angle recession lead to glaucoma

Clinical Features

- Deep anterior chamber
- Gonioscopy shows widened ciliary body band
- Raised IOP and glaucomatous optic nerve changes
- Other features of trauma (iridodialysis)

12. Enumerate the prognostic factors in a chemical eye injury. (3 marks)

Prognostic Factors

- The pH of the solution—strongly acidic (low pH) and strong alkali (very high pH)
- The duration of contact of the caustic substance with the ocular surface
- The percentage of the cornea is involved
- The amount of conjunctiva involved
- Other associated nonchemical injuries (like blunt or penetrating injury)
- Percentage of the limbal involvement (due to loss of limbal stem cells)

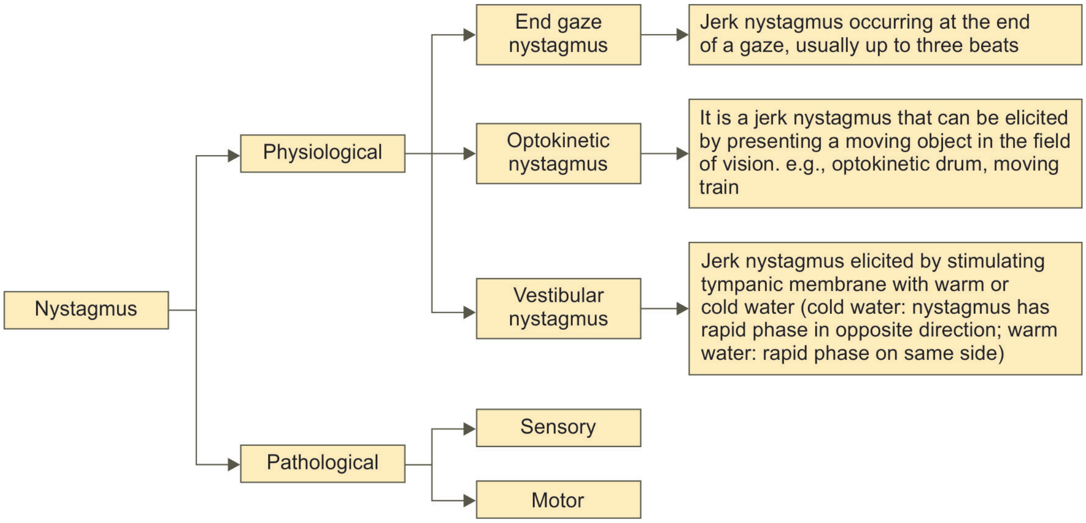
SHORT ESSAYS (MISCELLANEOUS)

1. Define nystagmus. Classify nystagmus. Describe the features of congenital nystagmus syndrome. (1+2+2 marks)

Nystagmus

Unilateral/bilateral, symmetric/asymmetric regular and rhythmic involuntary to and fro movement of eyes

Classification of Nystagmus


Nystagmus
Physiological
Pathological
End gaze nystagmus
Optokinetic nystagmus
Vestibular nystagmus
Sensory
Motor
Jerk nystagmus occurring at the end of a gaze, usually up to three beats
It is a jerk nystagmus that can be elicited by presenting a moving object in the field of vision. e.g., optokinetic drum, moving train
Jerk nystagmus elicited by stimulating tympanic membrane with warm or cold water (cold water: nystagmus has rapid phase in opposite direction; warm water: rapid phase on same side)

Features of Congenital Nystagmus Syndrome

- X-linked recessive or autosomal dominant inheritance
- Is seen at 2–3 months of age and persists through life
- Bilateral, symmetrical, horizontal, and jerky nystagmus
- Dampens on convergence, in sleep
- May develop head posture
- A null position is usually present

2. Enumerate four causes and describe clinical features of sensory nystagmus.
(2 + 1 = 3 marks)

Causes for Sensory Nystagmus

1. Congenital cataract
2. Optic nerve hypoplasia
3. Foveal hypoplasia-associated with aniridia, ocular albinism
4. Leber's congenital amaurosis

Clinical Features

- It is pendular nystagmus, with equal components and equal amplitude on either side
- It occurs due to reduced vision at a very early age before foveation takes place

SHORT ANSWERS (MISCELLANEOUS)

1. Explain the following terms concerning nystagmus. **(1 mark each)**

A. Amplitude
B. Frequency
C. Null point

A. Amplitude

The amount of movement of the eyeball from the midline.

B. Frequency

The number of oscillations occurring per second.

C. Null Point

- The position of the eye in which nystagmus is minimal
- In acquired nystagmus, the patient adopts a head posture such that the eye is in null point

2. State one cause for each of the nystagmus. (1 mark each)

A. Upbeat nystagmus
B. Downbeat nystagmus
C. Convergence retraction nystagmus

A. Upbeat Nystagmus

It is vertical nystagmus seen in brainstem lesions.

B. Downbeat Nystagmus

It is vertical nystagmus seen in cerebellar lesions and Arnold Chiari malformations.

C. Convergence Retraction Nystagmus

On attempted upgaze, globe retracts and eyes show nystagmus with a fast component towards the medial side (Perinaud's dorsal midbrain syndrome) caused by pinealomas and aqueductal stenosis.

3. Describe three causes of physiological nystagmus. (1 mark each)

1. **End-gaze nystagmus**
   - Jerk nystagmus occurring at the end of a gaze, usually up to three beats.
2. **Optokinetic nystagmus**
   - It is jerk nystagmus that can be elicited by presenting a moving object in the field of vision
   - For example, optokinetic drum, moving train
3. **Vestibular nystagmus**
   - Jerk nystagmus is elicited by stimulating the tympanic membrane with warm or cold water (cold water: nystagmus has a rapid phase in the opposite direction; warm water: rapid phase on the same side)

4. Enumerate three nystagmoid movements with the site of the lesion. (3 marks)

Nystagmoid movements are nystagmus-like movements of the eyes that do not qualify to be nystagmus

1. **Ocular flutter**
   - Horizontal oscillation and inability to fixate, seen in cerebellar pathology
2. **Opsoclonus**
   - Horizontal, vertical, and torsional oscillations associated with myoclonic movements of the face, seen in encephalitis
3. **Ocular bobbing**
   - Rapid downward movement of both eyeballs with a slow updrift
   - It is seen in pontine pathologies.

Multiple Choice Questions

COMPETENCY

OP1.1

1. The wavelengths of the EM radiation are absorbed by the cornea:

A. <300 nm
B. 300–350 nm
C. 350–400 nm
D. >750 nm

2. The maximum absorption spectrum for rhodopsin is:

A. 300 nm
B. 400 nm
C. 500 nm
D. 600 nm

3. Arrange these in the order of formation in the visual cycle:

(a) Lumirhodopsin
(b) Metarhodopsim I
(c) Al transretinal
(d) Bathorhodopsin

A. d, a, b, c
B. c, b, a, d
C. d, c, a, b
D. a, b, d, c

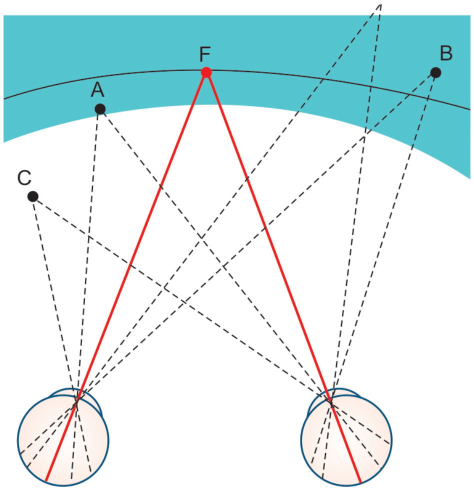
4. Match the following regarding development of vision:

a. At birth	i. Depth perception
b. 6–8 weeks	ii. Follows moving objects
c. 3 months	iii. Momentary fixation
d. 5–6 months	iv. Fixing on objects steadily

A. a - i, b - ii, c - iii, d – iv
B. a – iii, b –iv, c – ii, d – i
C. a – ii, b – iii, c – i, d – iv
D. a – iii, b – ii, c – i, d – iv

5. Identify the shaded region

A. Visual field
B. Horopter
C. Panum's fusional area
D. Blind spot



1. A 2. C 3. A 4. B 5. C

COMPETENCY

OP1.2

6. A high diabetic (RBS – 450 mg/dl) presents with a sudden blurring of vision. On examination, visual acuity is correctable to 6/6, V6 with –4.5D Sphere. Pupillary reactions and fundus are normal. The next logical step is to:

A. Perform MRI brain
B. Fundus fluorescein angiogram
C. ERG
D. Correct blood sugars

7. The refractive error that results due to a decrease in depth of the anterior chamber is:

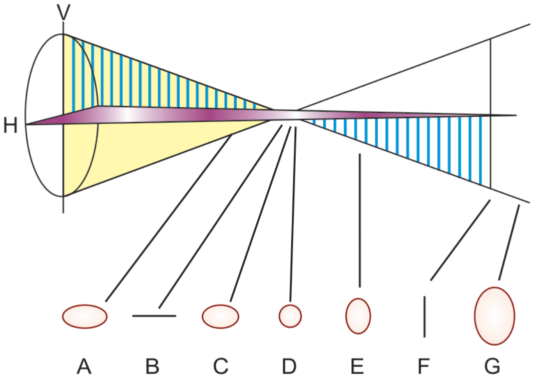
A. Myopia
B. Hypermetropia
C. Astigmatism
D. Presbyopia

8. All the following fundus features are associated with degenerative myopia *except:*

A. Posterior staphyloma
B. Lacquer cracks
C. Choroidal neovascular membrane
D. Choroidal folds

9. Identify the type of astigmatism when the retina is at point B in the following image:

A. Simple myopic astigmatism
B. Simple hypermetropic astigmatism
C. Emmetropia
D. Compound myopic astigmatism



10. The type of refractive error commonly seen in keratoconus is:

A. Hypermetropia
B. Hypermetropic astigmatism
C. Myopia
D. Myopic astigmatism

11. If a 56-year-old man underwent a small incision cataract surgery. A 6 mm long incision was made at the superior limbus. The type of astigmatism induced when the wound heals is:

A. Minus at 180°
B. Minus at 90°
C. Minus at 45°
D. Minus at 135°

12. The instrument used to measure corneal curvature is:

A. Pachymeter
B. Keratometer
C. A-scan
D. B-scan

13. A 12-year-old boy has –1DS in RE and –4.0DS in the left eye, but visual acuity in both eyes is correctable to 6/6, N6. The diagnosis is:

A. Strabismus
B. Amblyopia
C. Anisometropia
D. Anisekonia

6. D 7. B 8. D 9. B 10. D 11. B 12. B 13. C

14. If the corneal curvature is decreased from 50D to 43D, the refractive error induced is:

A. Myopia
B. Astigmatism
C. Hypermetropia
D. Presbyopia

COMPETENCY

OP1.3

15. When the beam of retinoscopy is moved from up to down, and you find the reflex moving upwards. The inference is:

A. Myopia <1D
B. Myopia >1D
C. Emmetropia
D. Hypermetropia

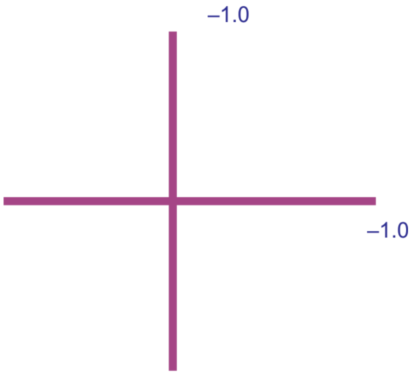
16. The instrument that automatically gives refraction values of the eye is:

A. Keratometer
B. Pachymeter
C. Confocal microscope
D. Autorefractometer

17. Scissoring reflex is seen in:

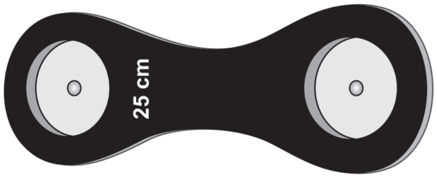
A. Keratoconus
B. Corneal scar
C. Corneal edema
D. Corneal dystrophy

18. Deduce the refractive power by the values of dry refractive performed at a distance of 2/3rd meter:



A. 0
B. –2.5 DS
C. –1.0 DS
D. –1.0 DS –1.0 Dcylinder at 90 degree

19. Identify the instrument:



A. Maddox rod
B. Bagolini's glasses
C. Mirror retinoscope
D. Krimsky's prism

20. Match the following regarding cycloplegics used in pediatric refraction:

a. Atropine	i. Has minimal cycloplegia
b. Homatropine	ii. 4–8 years
c. Cyclopentolate	iii. Infants
d. Tropicamide	iv. 1–4 years

A. a – i , b – iii , c – ii , d – iv
B. a – iii , b – iv , c – ii , d – i
C. a – ii , b – iii , c – i , d – iv
D. a – iv , b – iii , c –ii , d – i

14. C 15. B 16. D 17. A 18. B 19. C 20. B

COMPETENCY

OP.1.4

21. All of the following are corneal refractive procedures *except:*

A. Photorefractive keratectomy
B. LASIK
C. Small incision lenticule extraction
D. Corneal topography

22. The preferred techniques of correction of myopia of –6D and a thin cornea are:

A. Refractive lens exchange
B. Phakic IOL
C. LASIK
D. Small incision lenticule extraction

23. All of the following are performed before corneal ablation procedures *except:*

A. Tests for dry eye
B. Corneal topography
C. Fundoscopy
D. B-scan

24. Pick the true statement about excimer laser:

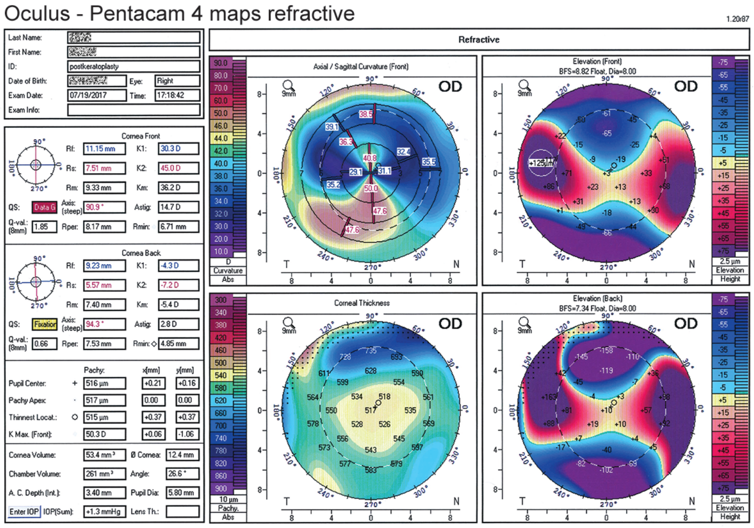
A. Derived from the two words “excited monomer”
B. The wavelength is 190 nm
C. Reaches the retina, there is a chance of retinal burn
D. Is the same as femtosecond laser

25. An 18-year-old girl underwent LASIK for right eye for –12D. After 6 months she developed progressive blurring of vision. On examination, there was a forward bulging of the cornea. The probable diagnosis is:

A. Post-LASIK ectasia
B. Post-LASIK keratoconus
C. Post-LASIK lenticonus
D. Post-LASIK staphyloma

26. Identify the corneal investigation

A. Pachymetry
B. A-scan
C. Topography
D. Confocal microscopy


Oculus - Pentacam 4 maps refractive

Last Name: [redacted]
First Name: [redacted]
ID: postkeratoplasty
Date of Birth: [redacted] Eye: Right
Exam Date: 07/19/2017 Time: 17:18:42
Exam Info:

Cornea Front
Rf: 11.15 mm K1: 30.3 D
Rs: 7.51 mm K2: 45.0 D
Rm: 9.33 mm Km: 36.2 D
QS: Data G Axis: (steep) 90.9 ° Astig: 14.7 D
Q-val.: (8mm) 1.85 Rper: 8.17 mm Rmin: 6.71 mm

Cornea Back
Rf: 9.23 mm K1: -4.3 D
Rs: 5.57 mm K2: -7.2 D
Rm: 7.40 mm Km: -5.4 D
QS: Fixation Axis: (steep) 94.3 ° Astig: 2.8 D
Q-val.: (8mm) 0.66 Rper: 7.53 mm Rmin: ◇ 4.85 mm

		Pachy:	x[mm]	y[mm]
Pupil Center:	+	516 µm	+0.21	+0.16
Pachy Apex:	·	517 µm	0.00	0.00
Thinnest Locat.:	○	515 µm	+0.37	+0.37
K Max. (Front):		50.3 D	+0.06	-1.06

Cornea Volume: 53.4 mm³ Ø Cornea: 12.4 mm
Chamber Volume: 261 mm³ Angle: 26.6 °
A. C. Depth (Int.): 3.40 mm Pupil Dia: 5.80 mm
Enter IOP IOP(Sum): +1.3 mmHg Lens Th.:

COMPETENCY

OP1.5

27. Pick the FALSE statement about the definition of amblyopia:

A. Visual acuity <6/12
B. RAPD is always present
C. Can be due to congenital cataract
D. Can be due to anisometropia

28. TRUE about principles of treatment of amblyopia:

A. Try to remove the amblyogenic cause
B. VEP can be used for follow–up
C. Squint should be treated first
D. Patching of the affected eye should be throughout the day

29. A 4-year-old boy has anisometropic amblyopia in his right eye. What is the regimen of patching of the unaffected eye?

A. 2 h/d, 2d/w
B. 2 h/d, 4 days patch followed by 1 day of non-patching
C. 2 h/d, 4 days patch followed by 4 days of non-patching
D. 4 hours/day, 4 days per week

30. All of these are modalities for the treatment of amblyopia *except:*

A. Using ground glass
B. Penalization
C. Occlusion
D. Tarsorrhaphy

31. Match the following regarding anisometropic amblyopia caused by a difference in refractive powers:

a. Myopic	i. 2D difference
b. Hypermetropia	ii. 1..5D difference
c. Astigmatism	iii. 4D difference

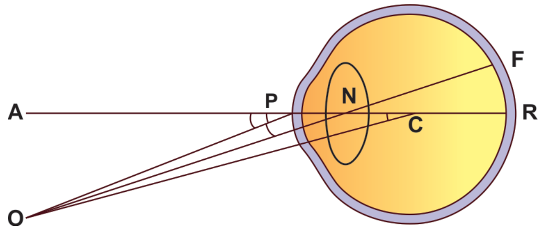
A. a – i, b – ii, c – iii
B. a – ii, b – iii, c – i
C. a – iii, b- i, c – ii
D. a – ii, b – i, c – iii

COMPETENCY

OP1 MISCELLANEOUS

32. Angle ONA in the picture represents: _____ angle.

A. Alpha
B. Beta
C. Gamma
D. Delta



33. The total power of the eye according to Listing's eye is:

A. 55 D
B. 58.2 D
C. 59.8 D
D. 60D

27. B 28. A 29. B 30. D 31. C 32. A 33. B

34. Negative angle kappa is seen in:

A. Hypermetropia
B. Astigmatism
C. Myopia
D. Presbyopia

COMPETENCY

OP2.1

35. All are the features of congenital ptosis *except:*

A. Usually myogenic
B. Ptosis is lesser in downgaze
C. Always bilateral
D. Requires surgery on an urgent basis if covering the pupil

36. Pick the TRUE statement about allergic lid edema:

A. Always associated with erythema
B. Always bilateral
C. It may be associated with visual loss
D. It may be associated with chemosis

37. A 23-year old woman presents with a hordeolum externum with a pus point. Treatment of choice is:

A. Incision and curettage
B. Epilation
C. Leave it alone
D. Systemic antibiotics

38. Which of the following can lead to preseptal cellulitis?

A. Hordeolum
B. Dacryocystitis
C. Conjunctivitis
D. All of the above

39. Which of the following tests are unnecessary for recurrent hordeola?

A. Blood sugars
B. Refraction
C. Schirmer's test
D. Nasal swab

40. The orientation of the incision while draining a chalazion on the conjunctional side is:

A. Horizontal, at the opening of Meibomian glands
B. Horizontal, at the upper edge of the tarsal plate
C. Vertical, along the gray region of the chalazion
D. Vertical, in the lower part of chalazion

41. Select the points in the clinical examination which are in favor of orbital cellulitis:

A. Lid edema
B. Lid erythema
C. Proptosis
D. Ophthalmoplegia
E. Chemosis

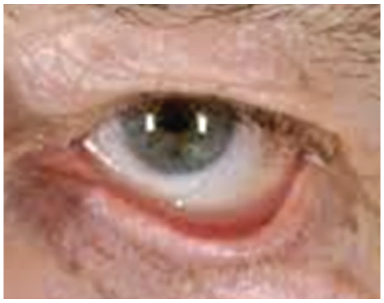
42. A 9-day-old child was brought by his parents with a history of watering from the right eye. After examination, it is concluded that he has CNLDO. What is the Rx of choice is this point?

A. Crigler massage
B. Probing
C. Stenting
D. Dacryocystorhinosotomy

34. C 35. C 36. D 37. B 38. D 39. C 40. C 41. C, D, E 42. A

43. A 50-year-old woman presents with epiphora for 3 years with on and off episodes of mucoid discharge in her right eye. The most probable diagnosis is:

A. Corneal ulcer
B. Chronic dacryocystitis
C. Chronic dacryoadenitis
D. Sjögren's syndrome



44. Identify the clinical condition

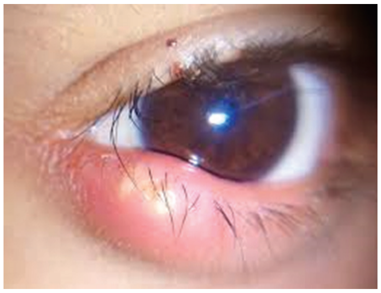
A. Entropion
B. Ectropion
C. Trichiasis
D. Ptosis

COMPETENCY

OP2.2

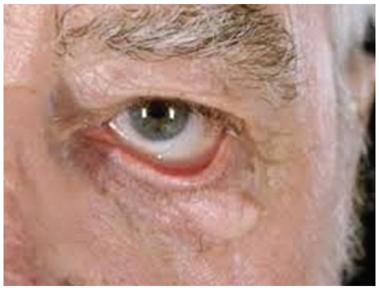
45. True about ptosis measurements:

A. MRD 1 + MRD 2 = LPS action
B. PFH – MRD2 = MRD 1
C. LPS action + MRD 1 = PFH
D. MRD 1 – MRD 2 = PFH



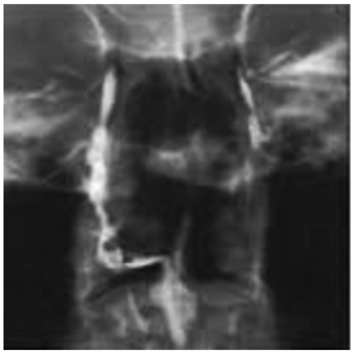
46. Identify the stage of hordeolum:

A. Cellulitis
B. Suppurative phase
C. Abscess
D. Fistula



47. Grade the below-shown ectropion:

A. Grade I
B. Grade II
C. Grade III
D. Grade IV

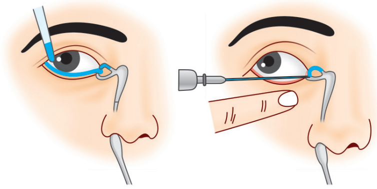


48. Which is the level of the block based on the dacryocystography image?

A. Punctum
B. Upper end of the sac
C. Lower end of the sac
D. Nasolacrimal duct

43. B 44. B 45. B 46. B 47. D 48. C

49. The results of the Jones test are shown below. Identify the cause of epiphora:



A. Canalicular block
B. Block in the lacrimal sac
C. Functional sac failure
D. Normal functioning lacrimal drainage

COMPETENCY

OP2.3

50. While syringing through the lower punctum, there is regurgitation from the upper punctum. All are possible sites of the block *except:*

A. Canaliculus
B. Common canaliculus
C. Lacrimal sac
D. Nasolacrimal duct

51. A hard stop on lacrimal probing is seen in:

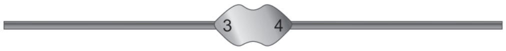
A. Canalicular block
B. Lacrimal sac block
C. Bony overgrowth
D. Nasolacrimal duct block

52. Match the names of instruments used in Lacrimal pathologies

a. Nettleship	i. Lacrimal probe
b. Bowman	ii. Punctum dilator
c. Lester Jones	iii. Tube

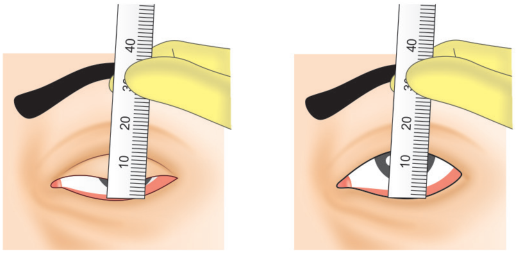
A. a – i, b – iii, c – ii
B. a – ii, b – i, c – iii
C. a – iii, b – ii, c – i
D. a – ii, b – iii, c – i

53. Identify the instrument



A. Punctum dilator
B. Jones tube
C. Lacrimal probe
D. None

54. Identify the test



A. PFH measurement
B. MRD measurement
C. Levator excursion
D. Proptosis measurement

55. The solution used for routine lacrimal syringing is:

A. Distilled water
B. Normal saline
C. Dextrose 5%
D. Sodium fluorescein

49. B 50. A 51. B 52. B 53. C 54. C 55. B

COMPETENCY

OP2.4

56. All of the following can cause visual loss in orbital cellulitis *except:*

A. Retrobulbar optic neuritis B. Compressive optic neuropathy
C. Exposure keratopathy D. Cystoid macular edema

57. A 50-year-old man underwent retinal detachment surgery with scleral buckling. On day three, he complained of severe pain, lid edema, congestion of conjunctiva, chemosis, and was found to have restricted elevation and depression. The most probable diagnosis is:

A. Orbital cellulitis B. Preseptal cellulitis
C. Optic neuritis D. Graft rejection

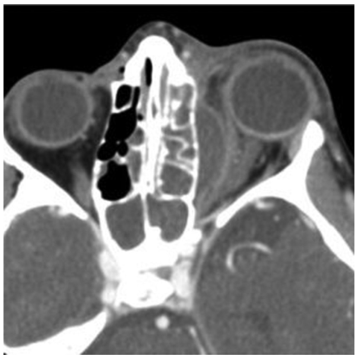
58. All of the following is a feature of orbital cellulitis on CT scan *except:*

A. Proptosis
B. Dilated superior ophthalmic vein
C. Soft tissue swelling posterior to the orbital septum
D. Soft tissue enhancement anterior to the orbital septum

59. Which of these clinical features suggests the development of a subperiosteal abscess in a case of orbital cellulitis?

A. Eccentric proptosis
B. Chemosis
C. Features of optic neuropathy
D. Exposure keratopathy

60. Identify the following image:



A. Orbital abscess
B. Orbital foreign body
C. Orbital periostitis
D. Cavernous sinus thrombosis

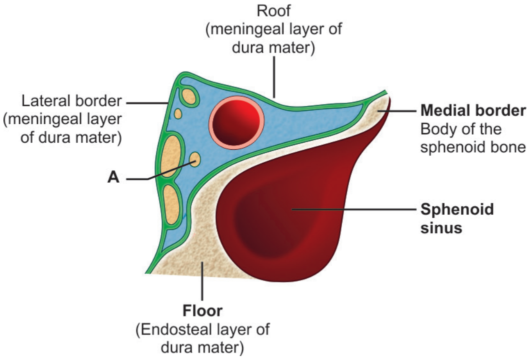
COMPETENCY

OP2.5

61. The characteristic MRI features of cavernous sinus thrombosis include:

A. Proptosis
B. Increased size of superior ophthalmic vein
C. Increased intensity of LR
D. Increased size of the cavernous sinus

62. Identify the structure marked by 'A':


Roof
(meningeal layer of
dura mater)
Lateral border
(meningeal layer
of dura mater)
Medial border
Body of the
sphenoid bone
A
Sphenoid
sinus
Floor
(Endosteal layer of
dura mater)

A. Oculomotor nerve
B. Trochlear nerve
C. Abducens nerve
D. Trigeminal nerve

56. D 57. A 58. B 59. A 60. A 61. D 62. C

63. These clinical features distinguish cavernous sinus thrombosis from orbital apex syndrome *except:*

A. Systemic features like fever
B. Bilaterality
C. Involvement of abducens nerve
D. Involvement of maxillary nerve

COMPETENCY

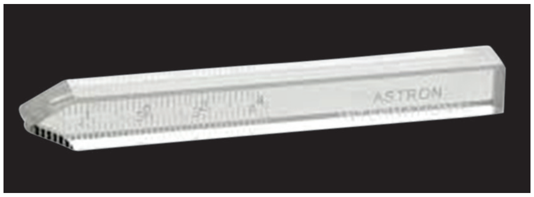
OP2.6

64. Which of these is a cause for eccentric proptosis with globe pushed inferonasally?

A. Optic nerve glioma
B. Optic nerve sheath meningioma
C. Pleomorphic adenoma of lacrimal gland
D. Graves's ophthalmopathy

65. All of these are capable of causing acute proptosis *except:*

A. Orbital hemorrhage
B. Orbital fractures with displaced bony fragment
C. Orbital emphysema
D. Blow-out fracture



66. Identify the instrument shown that is used in proptosis measurement

A. Luedde's exophthalmometer
B. Hertel's exophthalmometer
C. Henning's exophthalmometer
D. Kenneth's exophthalmometer

67. The most common cause of bilateral proptosis in adults is:

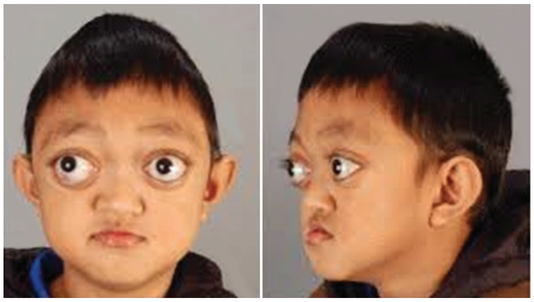
A. Cavernous sinus syndrome
B. Secondaries from neuroblastoma
C. Graves's ophthalmopathy
D. Histiocytosis

68. Match the following:

a. Lateral displaced eccentric proptosis	i. Lacrimal gland tumor
b. Medial displaced eccentric proptosis	ii. Maxillary sinus tumor
c. Inferior displaced eccentric proptosis	iii. Ethmoid mucocele
d. Superior displaced eccentric proptosis	iv. Frontal sinus mucocele

A. a – iii, b – i, c – iv, d – ii
B. a – ii, b – i, c – iv, d – ii
C. a – i, b – iv, c – iii, d – ii
D. a – ii, b – iii, c – i, d – iv

69. Identify the craniofacial anomaly.



A. Axenfeld syndrome
B. Reiger syndrome
C. Crouzon syndrome
D. Tolosa–Hunt syndrome

63. C 64. C 65. D 66. A 67. C 68. A 69. C

COMPETENCY

OP2.7

70. The most common primary malignancy of the orbit in childhood is:

A. Neuroblastoma B. Nephroblastoma
C. Rhabdomyosarcoma D. Burkitt's lymphoma

71. The malignancy of orbit that presents with Raccoon eyes is:

A. Retinoblastoma B. Rhabdomyosarcoma
C. Nephroblastoma D. Neuroblastoma

72. Which of the following orbital tumors increase in size during crying?

A. Orbital varix B. Cavernous hemangioma
C. Optic nerve glioma D. Rhabdomyosarcoma

COMPETENCY

OP2.8

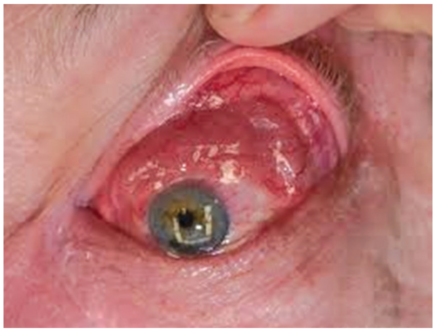
73. The tumor that 'hugs the globe' without invading it is:

A. Lymphoma B. Optic nerve glioma
C. Optic nerve sheath meningioma D. Teratoma

74. Tram–track calcification of optic nerve is classically seen in:

A. Optic nerve sheath meningioma B. Optic nerve glioma
C. Optic nerve sheath lymphoma D. Neuroblastoma

75. A 50-year-old man presents with gradual onset painless axial proptosis of the left eye. On examination, a rubbery mass is palpable under the orbital margin:



The most probable finding on MRI orbit is:

A. Tram–track appearance of the enlarged optic nerve
B. Fusiform enlargement of the optic nerve
C. Well encapsulated cystic mass with internal septations and fluid-filled levels
D. Homogeneous enhancement of soft tissue mass that indents the globe

76. A 3-year-old girl is brought with gradual onset proptosis of 2 weeks duration and upward displacement of the globe. A tumor was felt in the lower orbit and MRI revealed a tumor isodense with the extraocular muscle. The histopathology of the tumor is expected to have:

A. Well encapsulated tumors composed of large cavernous spaces containing RBCs
B. Large serous fluid-filled channels lined by endothelial cells
C. Astrocytic proliferation
D. Undifferentiated pluripotent mesenchymal elements, fibrovascular strands stacked with rhabdomyoblasts

70. C 71. D 72. A 73. A 74. A 75. D 76. D

77. Match the following orbital tumors with their corresponding histopathological features

a. Meningioma	i. Astrocytic proliferation
b. Glioma	ii. Serous fluid-filled channels
c. Lymphangioma	iii. Mixed variety of cell morphology
d. Pleomorphic adenoma	iv. Psammoma bodies

A. a – i, b – iii, c – ii, d – iv
B. a – iii, b – ii, c – i, d – iv
C. a – iv, b – i, c – ii, d – iii
D. a – iv, b – ii, c – i, d – iii

COMPETENCY

OP2 MISCELLANEOUS

78. The most common malignant lid tumor is:

A. Basal cell carcinoma
B. Squamous cell carcinoma
C. Sebaceous cell carcinoma
D. Melanoma

79. The most recent cause of epidemicity of orbital mucormycosis is:

A. Diabetes
B. HIV
C. Covid-19 infection
D. Use of antifungals

80. The sight-saving procedure in orbital compartment syndrome is:

A. Lateral tarsorrhaphy
B. Lateral canthotomy
C. Ocular bandaging
D. Bandage contact lens

81. The most common wall to be involved in blow-out fracture is:

A. Medial
B. Lateral
C. Roof
D. Floor

COMPETENCY

OP3.1

82. The type of discharge seen in an eye with bacterial conjunctivitis is:

A. Serous
B. Mucoid
C. Ropy
D. Stringy

83. The presence of brow-ache is seen in all *except:*

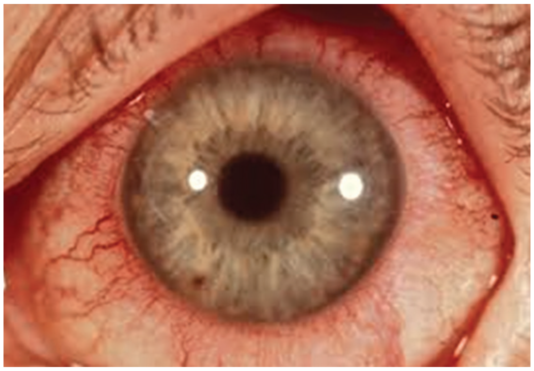
A. Conjunctivitis
B. Uveitis
C. Ciliary spasm
D. Raised IOP

84. The type of congestion seen in dry eye is:

A. Ciliary
B. Diffuse
C. Conjunctival
D. Interpalpebral

77. C 78. A 79. C 80. B 81. A 82. B 83. A 84. D

85. Identify the type of congestion:



A. Ciliary
B. Conjunctival
C. Diffuse
D. Interpalpebral

COMPETENCY

OP3.2

86. The part of the eye where ciliary tenderness is elicited is:

A. On cornea
B. At limbus
C. 2 mm behind the limbus
D. At insertions of recti muscles

87. A 33-year-old techie with red eye complains of foreign body sensation in both eyes towards the end of the day. On examination, there is interpalpebral congestion. Which of the following features point to a diagnosis of dry eye?

A. Decreased blink rate
B. Lower cornea staining by fluorescein
C. Increased Schirmer value
D. Decreased Tear film break-up time

88. All of the following are associated with follicular hypertrophy *except:*

A. Long-standing use of contact lens
B. Trachoma
C. Vernal conjunctivitis
D. Ophthalmia nodosa

COMPETENCY

OP3.3

89. The most common organism responsible for acute mucopurulent conjunctivitis:

A. *Staphylococcus aureus*
B. *Staphylococcus epidermidis*
C. *Streptococcus pyogenes*
D. Pneumococci

90. True about angular conjunctivitis:

A. Caused by *Mycoplasma*
B. Extremely infective
C. Affects angle of the anterior chamber
D. Source of infection is the nasal cavity

85. A 86. C 87. D 88. A 89. A 90. D

91. Match the following:

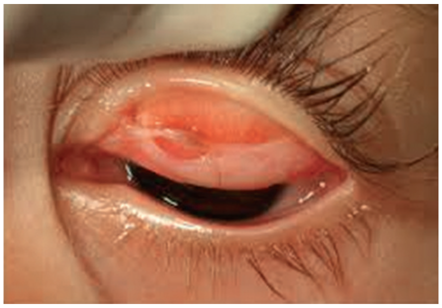
a. Pseudomembrauous	i. *Strep. hemolyticus*
b. Hyperacute	ii. *Moraxella*
c. True membranous	iii. *N. gonorrhea*
d. Angular	iv. *C. diphtheria*

A. a – i, b – ii, c – iii, d – iv
B. a – i, b – iii, c – iv, d – ii
C. a – iii, b – ii, c – i, d – iv
D. a – iv, b – iii, c – i, d – ii

92. A partially treated acute mucopurulent conjunctivitis leads to:

A. Chronic catarrhal conjunctivitis
B. Angular conjunctivitis
C. Adult inclusion conjunctivitis
D. Hyperacute conjunctivitis

93. Identify the conjunctival pathology:



A. Follicle
B. Papillae
C. Subconjunctival fibrosis
D. Membrane

COMPETENCY

OP 3.4

94. Condition in which papillae and follicle coexist is:

A. Allergic conjunctivitis
B. Mucopurulent
C. Trachoma
D. Adult inclusion

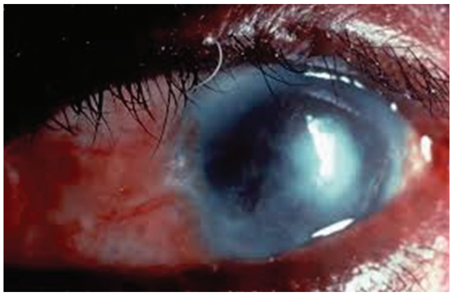
95. Systemic therapy for trachoma includes all the following *except:*

A. Oral tetracycline 250 mg QID × 4 weeks
B. Oral azithromycin 500 mg OD × 2 weeks
C. Oral erythromycin 250 mg QID × 4 weeks
D. Oral doxycycline 100 mg BID × 4 weeks

96. All of the following are corneal manifestations of trachoma *except:*

A. Pannus
B. Xerosis
C. Artl's line
D. Herbert's pits

97. Identify the stage of trachoma:



A. F
B. I
C. S
D. T
E. O

91. B 92. A 93. D 94. C 95. B 96. C 97. E

98. Which of these is FALSE about trachoma?

A. Primary infection is also called trachoma dubium
B. In the follicular stage, Leber cells are seen
C. *Chlamydia* is an obligate intracellular
D. McCallan's classification is followed presently

COMPETENCY

OP3.5

99. The best treatment for giant papillary conjunctivitis is:

A. Removal of the offending agent
B. Topical NSAIDs
C. Topical steroids
D. Intratarsal steroid injections

100. Match the following:

a. Contact lens	i. Atopic
b. Pseudogerontoxon	ii. Giant papillary
c. Tylosis	iii. Trachoma
d. Arlt's line	iv. Vernal keratoconjunctivitis

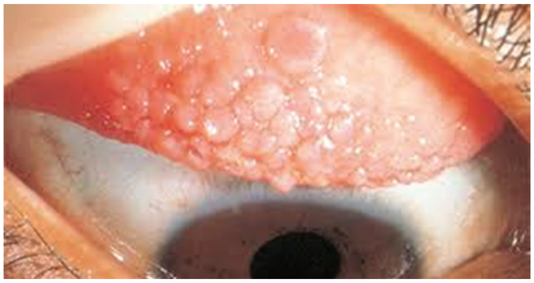
A. a–i, b – ii, c – iii, d–iv
B. a – iii, b – i, c – ii, d–iv
C. a – ii, b – iv, c – i, d – iii
D. a – iv, b– ii, c– iii, d– i

101. Horner–Tranta's dots are:

A. Epithelial erosions on the cornea
B. Raised whitish dots along the limbus
C. Follicles along limbus
D. Thickening of limbal tissue

102. Identify the conjunctival pathology:

A. Follicle
B. Papillae
C. Conjunctival cyst
D. None



103. Which of these is a mast cell stabilizer?

A. Sodium cromoglycate
B. Prednisolone
C. Acetylcysteine
D. Triamcinolone

98. D 99. A 100. C 101. B 102. B 103. A

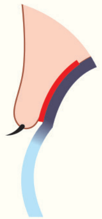
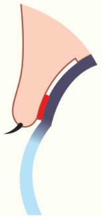
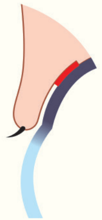
COMPETENCY

OP3.6

104. All are clinical implications of symblepharon *except:*

A. Diplopia
B. Lagophthalmos
C. Corneal xerosis
D. Uveitis

105. Match the following types of symblepharon:

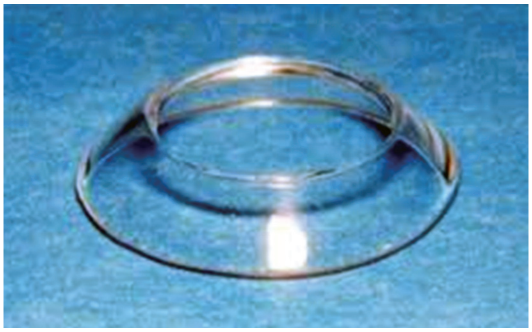
A. Anterior	i 
B. Posterior	ii 
C. Total	iii 

A. a: i, b: ii, c: iii
B. a: ii, b: iii, c: i
C. a: i, b: iii, c: ii
D. a: iii, b: ii, c: i

106. Adhesions between palpebral and bulbar conjunctiva are called:

A. Poliosis
B. Madarosis
C. Trichiasis
D. Symblepharon

107. Identify the following:



A. Keratoprosthesis
B. Contact lens
C. Symblepharon ring
D. Occlusive patch

104. D 105. B 106. D 107. C

COMPETENCY

OP3.7

108. The most accepted theory for the development of a pterygium is:

A. Malignant transformation of the conjunctiva
B. Benign hypertrophy of limbal stem cells
C. UV light-induced limbal stem cells loss leading to the growth of subconjunctival tissue
D. UV light-induced proliferation of fat cells

109. The concentration of mitomycin C used in pterygium excision is:

A. 0.02%
B. 0.2%
C. 2%
D. 2.0%

110. Which of these surgeries for pterygium excision is obsolete?

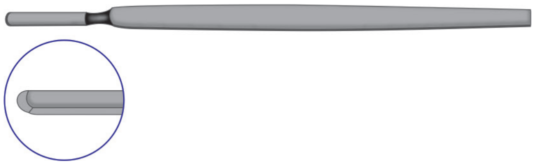
A. CLAU–Conjunctiva limbal autograft
B. Bare sclera
C. Use of mitomycin C
D. Mc Reynold's surgery

111. All of the following are degenerative conditions of conjunctiva *except:*

A. Pterygium
B. Pinguecula
C. Granuloma
D. Concretions

112. Identify the instrument used in pterygium excision:

A. Kimura's spatula
B. Desmarres's refractor
C. Tooke's knife
D. Foreign body spud

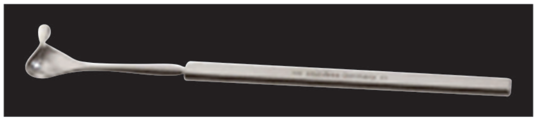


COMPETENCY

OP3.8

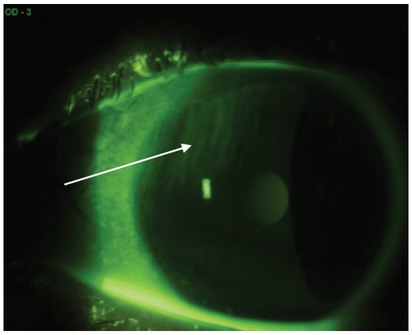
113. Identify the instrument:

A. Epilation forceps
B. Cat's paw
C. Foreign body spud
D. Desmarre's retractor



114. A 16-year-old biker complains of a sudden onset pricking sensation. On examination, fluorescein staining the following pattern is seen.
Where is the probable location of the foreign body?

A. Lower palpebral conjunctiva
B. Upper subtarsal conjunctiva
C. Fornix
D. Bulbar conjunctiva



108. C 109. B 110. C 111. B 112. C 113. D 114. A

115. Consequences of a conjunctival foreign body include all *except:*

A. Granuloma
B. Infection
C. Scleral perfection
D. Corneal xerosis

COMPETENCY

OP3.9

116. The letter 'G' prefixed to the drug for topical eye drops means:

A. Gropple
B. Gutta
C. Ground
D. Gruel

117. For a drug to be well absorbed through the ocular surface it should be:

A. Lipophilic only
B. Lipophobic only
C. Neither lipophilic nor lipophobic
D. Both lipophilic and lipophobic

118. Agents that decrease the surface tension help in cornea wetting. FALSE about this is:

A. Surfactants
B. Most commonly used chemical is BKC
C. Can cause dry eye in prolonged use
D. Decrease absorption of a drug

119. The normal capacity of the conjunctival cul-de-sac is:

A. 30 µl
B. 100 µl
C. 80 µl
D. 250 µl

COMPETENCY

OP4.1

120. Which layers of cornea regenerate without scarring?

A. Epithelium
B. Bowman's membrane
C. Descemet membrane
D. Endothelium

121. All of the following organisms can penetrate intact epithelium *except:*

A. *Neisseria gonorrhea*
B. *Meningococcus*
C. *Listeria*
D. *Chlamydia*

122. Keratocytes are derived from which layer of the cornea?

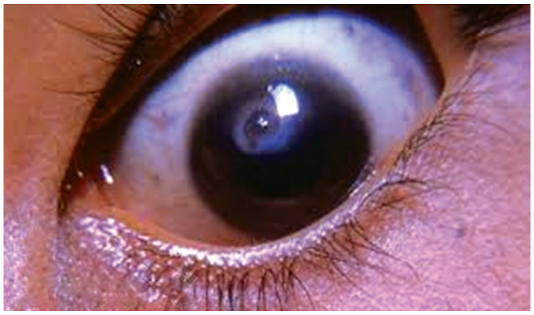
A. Epithelium
B. Bowman's membrane
C. Stroma
D. Endothelium

123. The cranial nerve affected in neurotrophic keratopathy is:

A. Oculomotor nerve
B. Optic nerve
C. Facial nerve
D. Trigeminal nerve

124. Identify the clinical condition:

A. Pseudocornea
B. Descemetocele
C. Perforation
D. Iris prolapse



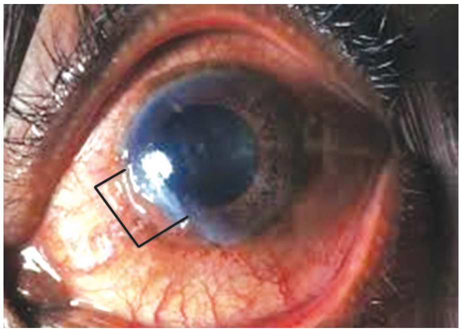
115. C 116. A 117. D 118. D 119. A 120. A,C 121. D 122. C 123. D 124. B

125. Following are the ways of treating a microperforation *except:*

A. Fibrin glue
B. Bandage contact lens
C. Corneal suturing
D. Pressure bandage

126. The most common systemic condition associated with the clinical image shown below:

A. Rheumatoid arthritis
B. Streptococcal infection
C. Acne rosaceae
D. SLE



COMPETENCY

OP4.2

127. Which of these features favor gram-positive organisms causing corneal ulcer?

A. Corneal ulcers always associated with hypopyon
B. More localized
C. Loss of corneal sensation
D. Affect peripheral cornea

128. Hypopyon corneal ulcer is typically caused by:

A. *Pseudomonas*
B. *Pneumococcus*
C. *Echinococcus*
D. *Meningococcus*

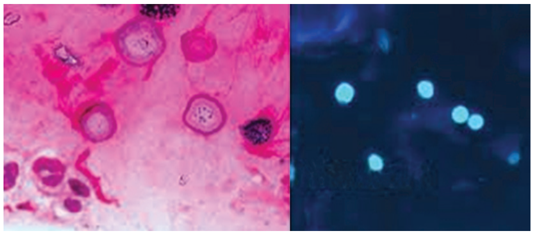
129. Match the following:

a. *Pseudomonas*	i. Satellite bodies
b. Yeast	ii. Dendrites
c. HSV	iii. Sloughing ulcer
d. *Acanthamoeba*	iv. Radial keratoneuritis

A. a – iii, b – i, c – iv, d – ii
B. a – iii, b – i, c – ii, d – iv
C. a – i, b – ii, c – iii, d – iv
D. a – iv, b – i, c – iii, d – ii

130. Identify the organism derived from corneal scrapings and stained with calcofluor stain:

A. Herpes simplex
B. *Acanthamoeba*
C. *Naegleria*
D. Herpes zoster



131. All of the following are manifestations of HSV in the eye *except:*

A. Dendritic ulcer
B. Pseudodendrites
C. Geographic ulcer
D. Disciform keratitis

125. C 126. A 127. B 128. B 129. B 130. B 131. B

COMPETENCY

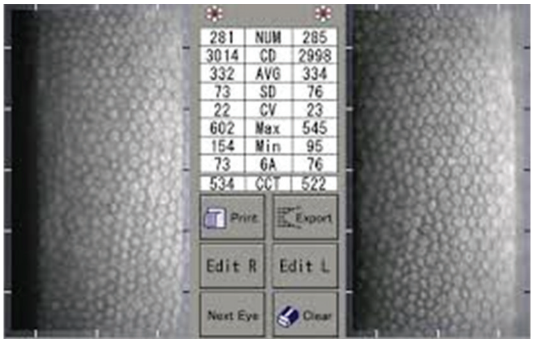
OP 4.3

132. True about corneal endothelium:

A. The number is constant throughout life
B. Polymegathism is an increase in the number of endothelial cells
C. Has Na^+–K^+–ATPase pumps
D. Has phagocytic activity

133. Identify the investigation:

A. Pachymetry
B. Specular microscopy
C. Keratometry
D. Photokeratoscopy



281	NUM	285
3014	CD	2998
332	AVG	334
73	SD	76
22	CV	23
602	Max	545
154	Min	95
73	6A	76
534	CCT	522

134. In presence of normal IOP and epithelial integrity, an endothelial count less than how much is associated with corneal edema?

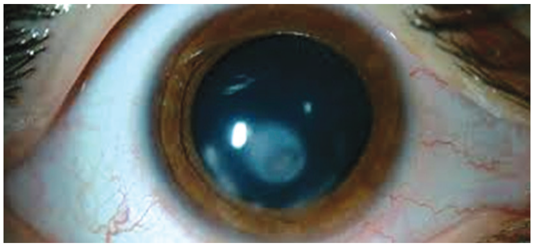
A. 3000 cells/mm^3
B. 2000 cells/mm^3
C. 1000 cells/mm^3
D. 750 cells/mm^3

135. All of the following are endothelial dystrophies *except:*

A. Fuch's
B. CHED
C. Lattice dystrophy
D. Posterior polymorphous dystrophy

136. Identify the clinical image:

A. Corneal ulcer
B. Corneal opacity
C. Corneal edema
D. Corneal vascularization



COMPETENCY

OP4.4

137. The normal tear film break-up time is:

A. 5–10s
B. 10–15s
C. 20s
D. 20–30s

138. All the following dyes are used for dry eye assessment *except:*

A. Rose Bengal
B. Fluorescein
C. Lissamine green
D. Giemsa stain

139. Which of the following invasive investigations helps in studying meibomian gland dysfunction?

A. Meibography
B. Ocular surface thermography
C. Meibomian angiography
D. Conjunctival impression cytology

132. C 133. B 134. D 135. C 136. B 137. B 138. D 139. A

140. Normal values of Schirmer test is:

A. 5–10 mm
B. 10–15 mm
C. 15–20 mm
D. 20–25 mm

141. All of these conditions predispose to dry eye *except:*

A. Allergic conjunctivitis
B. Bacterial conjunctivitis
C. Rosaceae keratoconjunctivitis
D. Blepharitis

142. When an eye with evaporative dry eye is stained with fluorescein the area of the cornea that gets stained is:

A. Central 3 mm
B. Along lid margins (superior and inferior limbus)
C. Interpalpebral area
D. Superior limbus

COMPETENCY

OP4.5

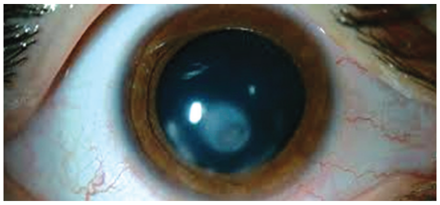
143. River blindness is caused by:

A. Loa loa
B. *Oncocerca volvulus*
C. *Echinococcus*
D. Tapeworm

144. The SAFE strategy is used to avoid corneal blindness due to:

A. Onchocerciasis
B. Vitamin A deficiency
C. Trachoma
D. Adenovirus

145. Grade the corneal opacity:



A. Nebula
B. Macula
C. Leucoma
D. Adherent leucoma

146. Keratomalacia is typically described concerning:

A. Corneal melt due to chemical injury
B. Corneal melt due to vitamin A deficiency
C. Corneal melt due to onchocerciasis
D. Corneal melt due to fungal keratitis

COMPETENCY

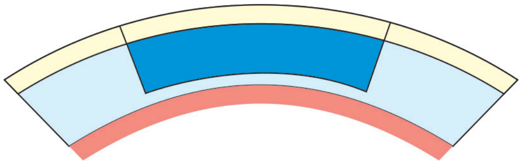
OP4.6

147. The preferred size of donor cornea should be more than the recipient bed by how many mm?

A. 0.25
B. 0.5
C. 1
D. 1.5

140. C	141. B	142. C	143. B	144. C	145. C	146. B	147. A

148. Identify the type of keratoplasty shown below:



A. Penetrating keratoplasty
B. Deep anterior lamellar keratoplasty
C. DSAEK
D. Keratoprosthesis

149. Kayes' dots are characteristic of:

A. Epithelial rejection
B. Stromal rejection
C. Subepithelial rejection
D. Endothelial rejection

150. The keratoprosthesis that uses a donor corneal button is:

A. Boston keratoprosthesis
B. Alfa-Cor
C. MOOKP
D. Stanford KPro

151. The wavelength of LASER used in phototherapeutic keratectomy:

A. 190–200 nm
B. 450 nm
C. 900 nm
D. 1200 nm

COMPETENCY

OP4.7

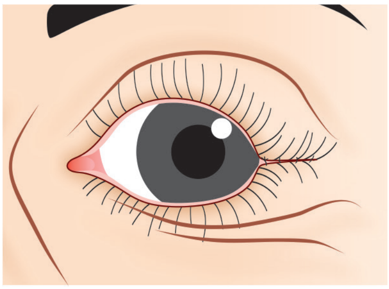
152. Early signs of exposure keratopathy due to facial palsy are seen in which part of the cornea?

A. Central
B. Upper
C. Lower
D. Whole cornea

153. All of the following are indications for tarsorrhaphy *except:*

A. Severe proptosis
B. Retrobulbar hemorrhage
C. Facial palsy
D. non-healing corneal ulcer

154. Identify the type of tarsorrhaphy:



A. Lateral
B. Bilateral
C. Paramedian
D. Median

COMPETENCY

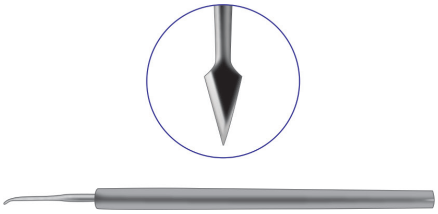
OP4.8

155. Which foreign body is associated with the formation of rust-ring?

A. Animal dander
B. Metal foreign body
C. Superglue
D. Wooden foreign body

148. B 149. B 150. A 151. A 152. C 153. B 154. A 155. B

156. Identify the instrument:

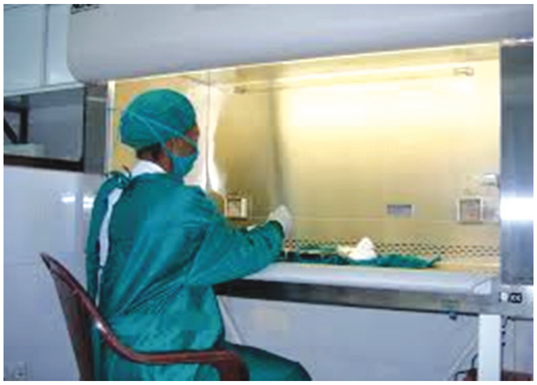


A. Toothed forceps
B. Nontoothed forceps
C. Spud
D. Needle

COMPETENCY

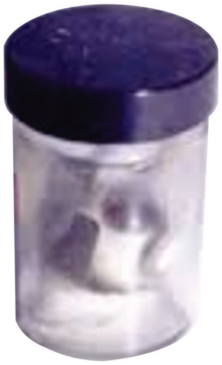
OP4.9

157. Identify the instrument:



A. Laminar flow
B. Moist chamber
C. Refrigerator
D. Cryopreservation

158. Identify the instrument:



A. Laminar flow
B. Moist chamber
C. Refrigerator
D. Cryopreservation

159. Which of these is an absolute contraindication for corneal donation?

A. Death due to MI
B. Cerebrovascular accident
C. Death due to an unknown cause
D. Death due to GI bleeding

160. All of the following are features of grade I donor corneal tissue *except:*

A. No corneal epithetical defects
B. Stroma is clear
C. Numerous Descemet's folds
D. No endothelial defects

161. FALSE about McCarey Kaufman medium:

A. Consists of Tc 199
B. HEPES buffer
C. Consists of chondroitin sulfate
D. For short-term storage

156. C 157. A 158. B 159. C 160. C 161. C

COMPETENCY

OP4.10

162. The period of time within which cornea has to be retrieved is:

A. 5 min of death	B. Half hour of death
C. 2 hours of death	D. 6 hours of death

163. The Transplantation of Human Organs Act was passed in:

A. 1992	B. 1993
C. 1994	D. 1995

164. Visual acuity remains normal in:

A. Keratoconus	B. Pellucid marginal degeneration
C. Posterior keratoconus	D. Keratoglobus

COMPETENCY

OP5.1

165. Management of nodular episcleritis includes all *except:*

A. Topical NSAIDs	B. Observation
C. Topical steroids	D. Surgical excision

166. The most common cause of episcleritis is:

A. Idiopathic	B. Rheumatoid arthritis
C. Pyoderma gangrenosum	D. Sweet syndrome

167. FALSE regarding episcleritis is:

A. Corkscrew vessels all around the lesion
B. It may be associated with uveitis
C. Can spread to underlying sclera
D. The vessels get blanched by topical phenylephrine

COMPETENCY

OP5.2

168. The most common cause of anterior scleritis is:

A. SLE	B. RA
C. Infectious	D. Idiopathic

169. SINS is typically seen after ________ days

A. 7	B. 21
C. 30	D. Anytime practically

170. All the following are signs of infectious scleritis *except:*

A. Unresponsive to steroids	B. Tend to be localized
C. Fever	D. Boggy and soft appearance

162. D 163. C 164. C 165. D 166. A 167. C 168. B 169. D 170. C

171. Melting of sclera without inflammation is characteristic of:

A. Scleromalacia perforans
B. Anterior nodular scleritis
C. Episcleritis
D. Posterior scleritis

172. All the following are features of scleritis *except:*

A. Usually associated with pain
B. Purplish due to vessels
C. Vessels are oriented crisscross
D. Requires a short course of steroids

COMPETENCY

OP6.1

173. In grade II cells, how many cells are counted in a window of 1 mm^3?

A. 1–5
B. 6–15
C. 16–25
D. 26–50

174. Nodules at the pupillary border are called:

A. Koeppe's nodules
B. Busacca's nodules
C. Kisch nodules
D. Behcet's nodules

175. The cataract associated uveitis is called:

A. Syndermatotic
B. Complicated
C. Nuclear
D. Christmas tree

176. The types of uveitis associated with an increase in IOP are all *except:*

A. Behcet syndrome
B. Zoster uveitis
C. Posner Schlossman
D. Pupillary block

177. The treatment of choice of panophthalmitis is:

A. Enucleation
B. Evisceration
C. Exenteration
D. Intravitreal antibiotic

COMPETENCY

OP6.2

178. All of the following are signs of acute uveitis *except:*

A. Nonpigmented KPs
B. Crenated KPs
C. Flare
D. Ciliary tenderness

179. All of these tends to be chronic in presentation *except:*

A. Phacolytic glaucoma
B. Sarcoid associated uveitis
C. Sympathetic ophthalmia
D. Juvenile idiopathic arthritis-associated uveitis

171. A 172. D 173. C 174. A 175. B 176. A 177. B 178. B 179. A

180. Which of the following is true?

A. Anterior synechiae develop due to uveitis
B. Posterior synechiae cannot be broken
C. The presence of synechiae in a cataract complicates the surgery
D. The presence of cataract in a case of uveitis always means inflammation is chronic

181. All of the following are complications of long-standing *except:*

A. Ciliary body shutdown
B. Band-shaped keratopathy
C. Phthisis bulbi
D. Hypopyon

COMPETENCY

OP 6.3

182. Heerfordt syndrome is characterized by all *except:*

A. Bilateral granulomatous uveitis
B. Parotid enlargement
C. Skin rashes
D. Erythema nodosum

183. In Vogt–Koyanagi–Harada syndrome, the type of retinal detachment is:

A. Exudative
B. Rhegmatogenous
C. Tractional
D. Combination of A and B

184. A 45-year-old patient presents with floaters and on fundus examination, there is a headlight in the fog appearance. Which of the following drugs is helpful?

A. Valganciclovir
B. Acyclovir
C. Sulfasalazine + pyrimethamine
D. Rifampicin

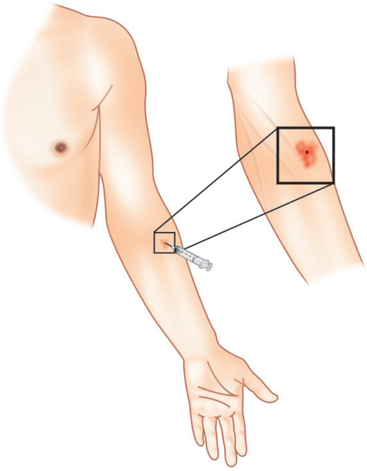
185. Match the following:

a. Tinu	i. Acute anterior uveitis
b. Sarcoidosis	ii. Hypertensive uveitis
c. Herpes zoster	iii. Granulomatous uveitis

A. a: iii, b: i, c: ii
B. a: i, b: iii, c: ii
C. a: ii, b: i, c: iii
D. a: i, b: ii, c: iii

186. A 27-year-old man presented with recurrent bilateral uveitis with aphthous ulcers. The following test was performed.

What is the most possible systemic association for uveitis?



A. Tuberculosis
B. Sarcoidosis
C. Behcet's syndrome
D. Herpes zoster

180. C 181. D 182. D 183. A 184. C 185. B 186. C

COMPETENCY

OP6.4

187. UGH syndrome is mainly described with respect to:

A. Blunt trauma
B. Penetrating trauma
C. Use of PCIOL
D. Use of ACIOL

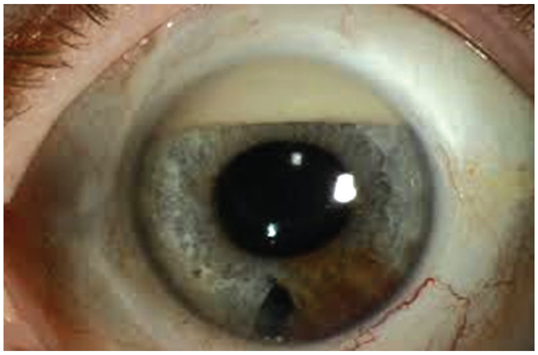
188. Which of the following uveitis is associated with spontaneous hyphema?

A. Intermediate uveitis
B. Fuch's heterochromic iritis
C. Anterior uveitis associated with Behcet's syndrome
D. Posterior uveitis associated with tuberculosis

189. All of the following are indications for paracentesis of hyphema *except:*

A. IOP >50 mm Hg for more than 48 hours
B. Endothelial blood staining
C. IOP >21 mm Hg for more than 2 days
D. IOP >30 mm Hg for more than 1 week

190. Identify the following image:



A. Hypopyon
B. Inverse hypopyon
C. Pseudohypopyon
D. Hyphema

191. All of the following are associated with a hypopyon *except:*

A. Endophthalmitis
B. Corneal ulcer
C. Uveitis
D. Corneal epithelial erosion

COMPETENCY

OP6.5

192. The angle of the anterior chamber is not visible on slit-lamp examination due to:

A. Total internal reflection
B. Refraction
C. Dispersion
D. Diffraction

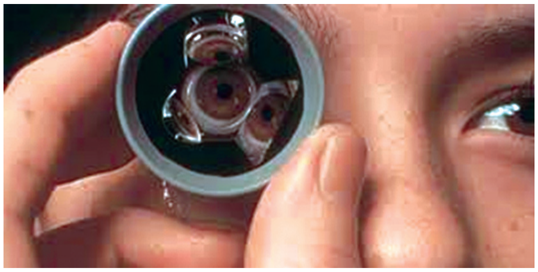
193. The structures seen in grade II Shaffer's system are:

A. Schwalbe's line
B. Schwalbe's line, trabecular meshwork
C. Schwalbe's line, trabecular meshwork, scleral spur
D. Schwalbe's line, trabecular meshwork, scleral spur, iris root

187. D 188. B 189. C 190. B 191. D 192. A 193. B

194. Arrange the following in the order from anterior to posterior as seen in gonioscopy

A. Root of iris, Schwalbe's line, ciliary body band, trabecular meshwork
B. Ciliary body band, trabecular meshwork, root of iris, Schwalbe's line
C. Root of iris, Schwalbe's line, trabecular meshwork, ciliary body band,
D. Schwalbe's line, trabecular meshwork. Root of iris, ciliary body band

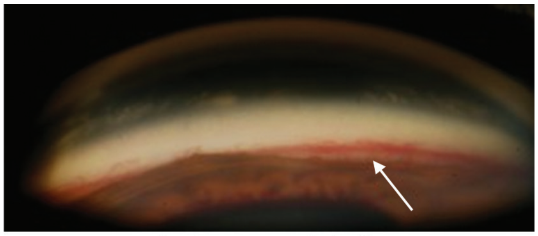


195. Identify the test:

A. Tonometry
B. Gonioscopy
C. LASER trabeculoplasty
D. Trabeculectomy

196. All may be a cause for the following *except:*

A. Diabetic retinopathy
B. Central retinal venous occlusion
C. Fuch's heterochromic iridocyclitis
D. Hypertensive retinopathy



Angle neovascularization

COMPETENCY

OP6.6

197. The most prominent structure visible on gonioscopy is:

A. Schwalbe's line
B. Trabecular meshwork
C. Scleral spur
D. Ciliary body band

198. Peripheral anterior synechiae are adhesions between:

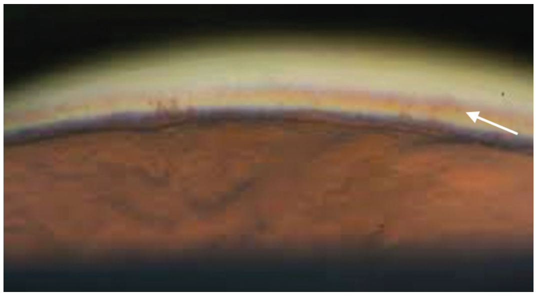
A. Iris and lens
B. Pupil and lens
C. Schwalbe's line and iris
D. Trabecular meshwork and Schwalbe's line

199. All of these cause blood in Schlemm's canal *except:*

A. Increased episcleral venous pressure
B. Trauma
C. Caroticocavernus fistula
D. Subconjunctival hemorrhage

200. Identify the structure in the image pointed with the arrow:

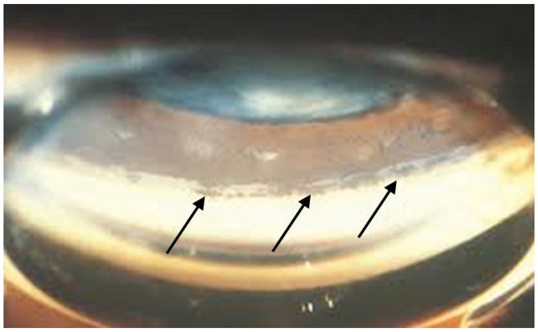
A. Schwalbe's line
B. Trabecular meshwork
C. Scleral spur
D. Iris root



194. D 195. B 196. D 197. B 198. C 199. D 200. B

201. **A 20-year-old male presented with a history of injury to the eyeball with a cricket ball. Gonioscopy was performed after hyphema resolved.**

The following picture is seen. Which of the following is TRUE about this?



A. He may develop glaucoma in future
B. He will not develop glaucoma
C. He will develop intractable absolute glaucoma
D. It has no relation to glaucoma

COMPETENCY

OP6.7

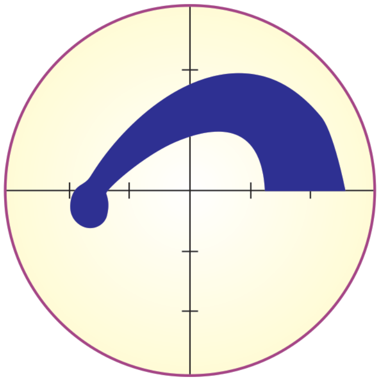
202. **The drug of choice for normal-tension glaucoma is:**

A. Timolol
B. Betaxolol
C. Carvedilol
D. Esmolol

203. **The cut-off value for diagnosing primary open-angle glaucoma based on IOP more than:**

A. 20 mm Hg
B. 21 mm Hg
C. 22 mm Hg
D. 23 mm Hg

204. **Identify the type of field defect:**



A. Siedel's scotoma
B. Superior arcuate scotoma
C. Inferior arcuate scotoma
D. Ring scotoma

205. **The classical retinal nerve fiber thinning in glaucoma as seen on optical coherence tomography is:**

A. Superior and inferior peripapillary thinning
B. Macular thinning
C. Nasal peripapillary and macular thinning
D. Nasal thinning only

206. **Buphthalmos is characterized by all *except:***

A. Cloudy cornea
B. Enlarged cornea
C. Increased axial length
D. Induced hypermetropia

201. A 202. B 203. B 204. B 205. A 206. D

COMPETENCY

OP6.8

207. Following tests are useful in sarcoidosis *except:*

A. Positive pathergy
B. Serum ACE levels
C. Serum calcium
D. Hilar lymphadenopathy on chest imaging

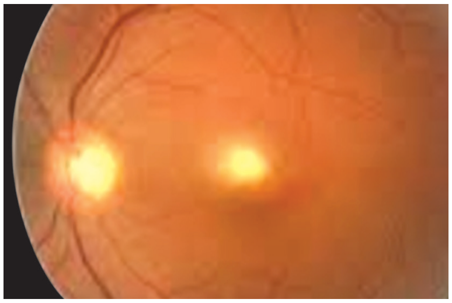
208. A 40-year-old patient with decreased sensorium has choroid tubercles in both eyes. Best investigation is:

A. Chest X-ray
B. MRI brain
C. Ultrasound abdomen
D. Doppler of carotids

209. Which of the following tests is not useful in intermediate uveitis?

A. MRI brain for multiple sclerosis
B. Chest X-ray for sarcoid
C. Serum ELISA for *Borrelia*
D. Anterior segment OCT

210. Which of the following investigations is helpful in this posterior uveitis?



A. IgM toxoplasmosis
B. Serum calcium
C. Mantoux test
D. Pathergy test

211. A 7-year-old child was brought by parents with occasional redness in one eye. On examination, posterior synechiae are seen in that eye with no other signs of inflammation. Which of these systemic investigations are helpful in this case?

A. HLA B27
B. Kidney biopsy
C. Serum ACE
D. ANA

COMPETENCY

OP6.9

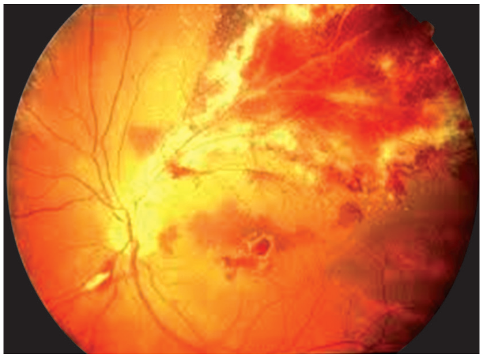
212. Atropine helps in uveitis in the following ways *except:*

A. Relaxes the ciliary muscle and decreases spasm
B. Pulls the iris away from the lens and reduces synechiae formation
C. Decreases the endothelial carbonic acid activity
D. Reduces the vascularity and inflammation

213. All of the following are immunosuppressants used in uveitis *except:*

A. Methotrexate
B. Cyclophosphamide
C. Tacrolimus
D. Nimesulide

207. A 208 B 209. D 210. A 211. D 212. C 213. D



214. The drug of choice used in this posterior uveitis is:

A. Ganciclovir
B. Acyclovir
C. Sulfasalazine
D. Trimethoprim

215. Which antiglaucoma is contraindicated in glaucoma associated with uveitis?

A. Prostaglandins
B. Beta-blockers
C. Alpha agonists
D. Carbonic anhydrase inhibitors

216. A postoperative patient complains of severe ocular pain and diminution of vision. A yellow reflex is seen in the pupil and a B-scan reveals exudates in the vitreous cavity. All of the following are advocated *except:*

A. Intravitreal vancomycin
B. Intravitreal ceftazidime
C. Topical steroids
D. Intravenous steroids

COMPETENCY

OP6.10

217. All the following are expected in a child with JIA *except:*

A. Phthisis bulbi
B. Corneal edema
C. Band-shaped keratopathy
D. Complicated cataract

218. Which of the following is required in treating HLA-B 27 associated uveitis to prevent recurrence?

A. Long-term cycloplegic
B. Immunosuppression
C. Systemic steroids
D. Short-term steroids

219. Which disease is described as the one causing 'cold hypopyon'?

A. VKH syndrome
B. Sarcoidosis
C. Behcet's syndrome
D. Toxoplasmosis

COMPETENCY

OP7.1

220. Identify the layers of the lens from outside to inside:

A. Capsule, cortex, adult nucleus, fetal nucleus
B. Cortex, capsule, adult nucleus, fetal nucleus
C. Fetal nucleus, adult nucleus, cortex, capsule
D. Capsule, cortex, fetal nucleus, adult nucleus

214. A 215. A 216. D 217. B 218. A 219. C 220. A

221. The thickest portion of the capsule of the lens is:

A. Anterior capsule
B. Posterior capsule
C. Pre-equatorial capsule
D. Equator

222. Pick the FALSE statement about the nucleus of the lens:

A. Hard
B. The oldest part of the lens
C. Water-insoluble
D. Water-soluble

223. The most abundant found proteins in the lens are:

A. Alpha crystallins
B. Beta crystallins
C. Gamma crystallins
D. Albuminoids

224. TRUE about the functions of the lens is:

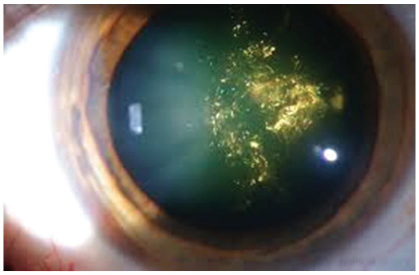
A. The refractive index is 1.30
B. A most important part of refractive media
C. Absorbs UV light
D. Vascular structure

COMPETENCY

OP7.2

225. Which of the following is a probable cause for this type of cataract?

A. Uveitis
B. Trauma
C. Senile
D. Diabetes



226. The term cataracta brunescens is referred to which type of cataract?

A. Cortical cataract
B. Nuclear cataract
C. Subcapsular cataract
D. Capsular cataract

227. A child is born with a membranous cataract. Which infection would you like to rule out?

A. Measles
B. Mumps
C. Diphtheria
D. Rubella

228. Match the following:

a. Christmas tree cataract	i. Diabetes
b. Snowflake cataract	ii. Galactosemia
c. Oil droplet cataract	iii. Wilson's disease
d. Sunflower cataract	iv. Myotonic dystrophy

A. a: iii, b: i, c: ii, d: iv
B. a: iv, b: i, c: ii, d: iii
C. a: i, b: ii, c: iii, d: iv
D. a: ii, d: iv, c: iii, d: ii

221. C 222. D 223. B 224. C 225. A 226. B 227. D 228. B

229. All of the following are features of Morgagnian cataract *except:*

A. Calcifications on the surface
B. Swollen lens
C. Sunken nucleus
D. Shallow anterior chamber

COMPETENCY

OP7.3

230. Which of the following is TRUE about biometry?

A. Calculation of the power of the IOL
B. A denotes axial length in SRK formula
C. Can be performed using A-scan only
D. The patient should be in general anesthesia

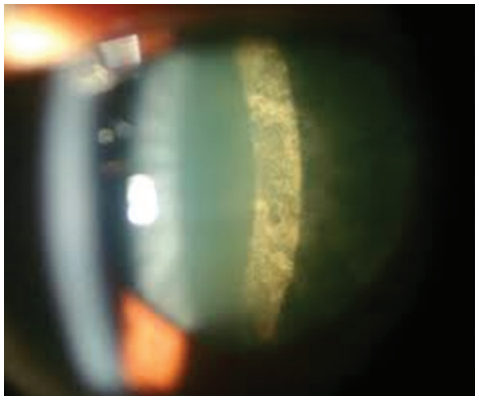
231. A diabetic patient with a cataract has a visual acuity of 6/60 in the affected eye and the lens shows grade I nuclear sclerosis. What is the next logical step?

A. Perform biometry
B. Post him for surgery
C. Rule out diabetic maculopathy
D. Perform B scan

232. Which of the following macular function tests are useful in a dense cataract and visual acuity of hand movements?

A. Color vision
B. Two-point discrimination
C. Photo-stress recovery test
D. Entoptic visualization

233. Identify the type of cataract:



A. Cortical cataract
B. Nuclear cataract
C. Posterior subcapsular cataract
D. Anterior capsular cataract

234. Match the following symptoms of cataracts:

a. Nuclear cataract	i. Induced hypermetropia
b. Cortical cataract	ii. Second sight
c. Posterior subcapsular	iii. Glare

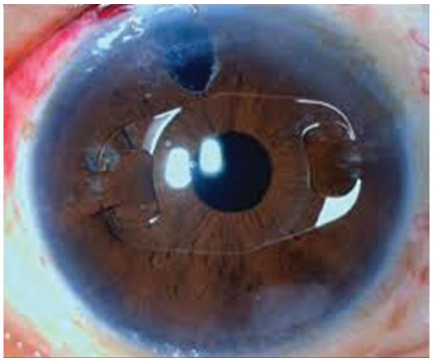
A. a: ii, b: i, c: iii
B. a: i, b: iii, c: ii
C. a: iii, b: i, c: ii
D. a: ii, b: i, c: iii

229. A 230. A 231. C 232. D 233. C 234. A

COMPETENCY

OP7.4

235. Identify the IOL:



A. ACIOL
B. PCIOL
C. Iris claw lens
D. Scleral fixated lens

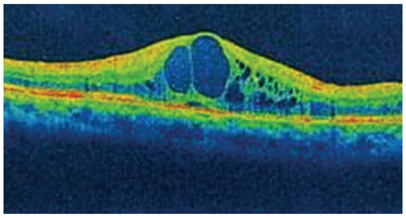
236. Which type of incision is associated with the least astigmatism in SICS?

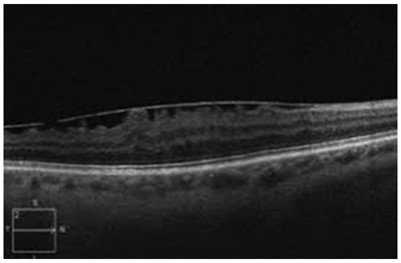
A. Smile
B. Frown
C. Straight
D. Circumlinear

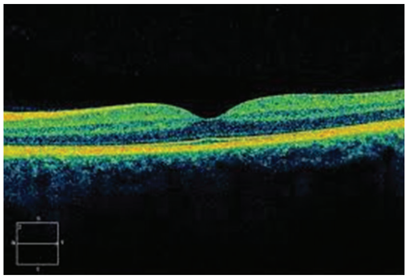
237. While performing a cataract extraction, during cortical aspiration, the anterior chamber becomes deep suddenly, and the pupil appears distorted in one angle. The most probable intraoperative complication is:

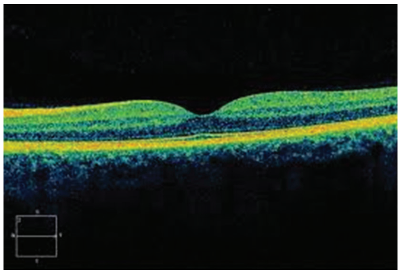
A. Posterior capsular rupture
B. Posterior capsular rupture with rupture of anterior hyaloid
C. Nucleus drop in vitreous
D. Expulsive choroidal hemorrhage

238. A 6 weeks postoperative cataract patient complains of metamorphopsia. On, examination, there is a honeycomb appearance at the macula. What is the probable pattern on OCT?

A.


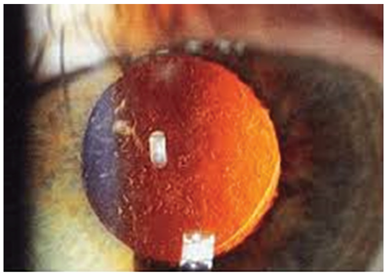
B.


C.


D.


235. C 236. B 237. B 238. A

239. What is the treatment of choice for this condition?



A. IOL explantation
B. YAG capsulotomy
C. Pan retinal photocoagulation
D. Surgical removal of the membrane

COMPETENCY

OP7.5

240. All of the features are seen following peribulbar anesthesia *except:*

A. Pupillary dilatation
B. Ophthalmoplegia
C. Loss of corneal sensation
D. Loss of red reflex

241. A patient posted for cataract surgery was given a retrobulbar block. He complains of severe pain in his eye. On examination, the eye is proptosed and very hard. No ocular movements are seen. Management of this patient includes all *except:*

A. Ocular massage
B. Postponement of surgery
C. Immediate cataract surgery
D. Intravenous mannitol to decrease IOP

242. All the following are true about the use of anesthetics in peribulbar anesthesia *except:*

A. Can cause an oculocardiac reflex
B. Can cause myotoxicity
C. Is given into the muscle cone
D. Causes loss of pupillary reflex

243. Pick the instructions that you would like to give a patient following cataract surgery:

A. Topical steroids
B. Topical antibiotics
C. Wash face and hair every day
D. Systemic antibiotics

244. Following measures are taken to prevent postoperative endophthalmitis while performing a cataract surgery *except:*

A. Use of antiglaucoma medications
B. Use of povidone-iodine to the ocular surface
C. Use of intracameral antibiotics
D. Use of eye shield

COMPETENCY

OP7.6

245. A 58-year-old patient with sinus bradycardia fell unconscious while administering a peribulbar block. What is the most probable diagnosis?

A. Ocular myotoxicity
B. Oculocardic reflex
C. Oculocephalic reflex
D. Vasovagal attack

239. B 240. D 241. C 242. C 243. A, B 244. A 245. B

246. A diabetic patient with grade II nuclear sclerosis has a visual acuity of 6/60. OCT of the macula shows cystoid spaces in the fovea. Which of the following is FALSE?

A. Perform LASER treatment for maculopathy first before cataract surgery
B. Perform cataract surgery first followed by LASER for maculopathy
C. Give intravitreal anti-VEGF at the time of surgery
D. Removal of cataract will heal maculopathy

247. An 80-year-old presents with a bilateral hypermature cataract. The fundus is not visualized. All of the following can be used to prognosticate visual recovery following cataract surgery *except:*

A. Color vision
B. Pupillary reactions
C. Projection of rays
D. Perception of light

248. Which of the following is an indication for cataract surgery?

A. Hypermature cataract only
B. Mature cataract only
C. Only cortical cataract
D. Any visually significant cataract

249. Which of the following is an absolute contraindication for cataract surgery?

A. HIV
B. Congenital rubella syndrome
C. Hepatitis C
D. None of the above

COMPETENCY

OP8.1

250. Hollenhorst plaques are made up of:

A. Amyloid
B. Cholesterol
C. Calcium
D. Atheroma

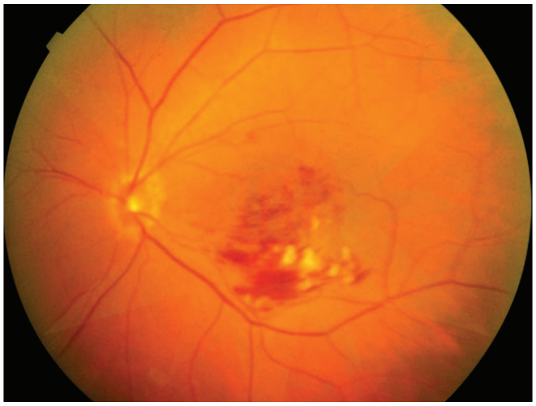
251. Cattle-trucking is characteristic of:

A. Central retinal artery occlusion
B. Central retinal vein occlusion
C. Macular vein occlusion
D. Ophthalmic artery occlusion

252. All of the following differentiates between central retinal artery occlusion and ophthalmic artery occlusion *except:*

A. Painless
B. Perception of light
C. Cherry red spot
D. Amalric sign

253. What is the diagnosis?



A. Ischemic CRVO
B. Non-ischemic CRVO
C. Papillophlebitis
D. Macular branch vein occlusion

246. A 247. A 248. D 249. D 250. B 251. A 252. A 253. D

254. Following features help in differentiating nonischemic from ischemic CRVO *except:*

A. Visual acuity
B. Number of hemorrhages
C. Number of capillary drop out areas on FFA
D. Presence or absence of disc edema

COMPETENCY

OP8.2

255. The parameters of LASER used in PRP is:

A. 500 µm size
B. 100 µm size
C. 50 µm size
D. Does not depend on the size

256. Match the following uses of LASER with its properties:

a. Proliferative retinopathy	i. Cuts of the blood supply
b. Retinal tumors	ii. Converts ischemic retina to anoxic retina
c. CSCR	iii. Causes adhesions between NSR and RPE
d. Retinal tears	iv. Increases the pump function of RPE

A. A: iii, b: i, c: ii, d: iv
B. a: ii, b: i, c: iv, d: iii
C. a: i, b: ii, c: iii, d: iv
D. a: ii, b: i, c: iii, d: iv

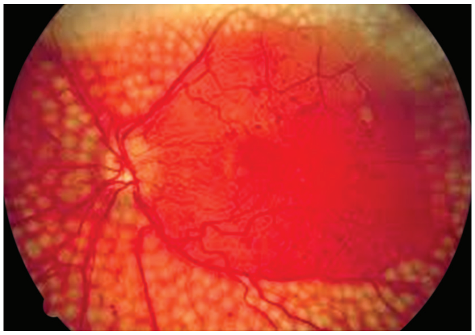
257. The type of LASER used in PRP is:

A. Excimer
B. Nd: YAG
C. Femtosecond
D. Argon

258. All of the following are complications of extensive LASER PRP *except:*

A. Rhegmatogenous retinal detachment
B. Exudative retinal detachment
C. Optic atrophy
D. Decrease in the visual field

259. Identify the lesion on the retina:



A. Choroiditis
B. Retinitis
C. Atrophic hole
D. Marks of photocoagulation

COMPETENCY

OP8.3

260. The image formed by an indirect ophthalmoscope is:

A. Real, inverted, and minified
B. Real, inverted, and magnified
C. Erect and magnified
D. Erect and minified

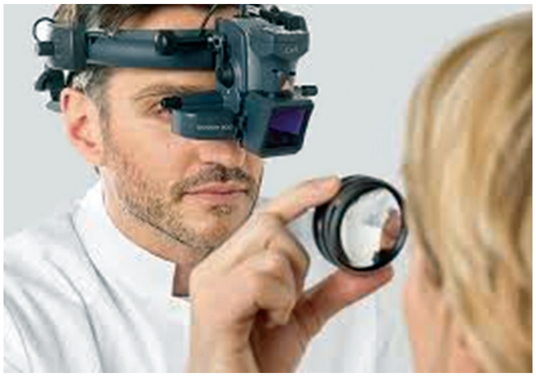
254. B 255. A 256. B 257. D 258. A 259. D 260. B

261. The advantage of direct ophthalmoscope over indirect ophthalmoscope is:

A. The image is more magnified
B. The image is erect and not reversed
C. It is easier to master
D. All of the above

262. All of the following lenses can be used in the slit-lamp biomicroscopy *except:*

A. +90D
B. +78D
C. +30D
D. –58D



263. Identify the instrument:

A. Indirect ophthalmoscope
B. Direct ophthalmoscope
C. Gonioscope
D. Specular microscope

264. Match the following:

a. Direct ophthalmoscope	i. Real, inverted and 3x
b. Indirect ophthalmoscope	ii. Real inverted, 0.9x
c. Slit lamp biomicroscopy	iii. Erect, magnified 15x

A. a: i, b: iii, c: ii
B. a: iii, b: i, c: ii
C. a: i, b: ii, c: iii
D. a: iii, b: ii, c: i

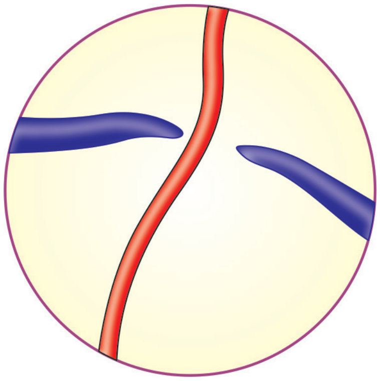
COMPETENCY

OP8.4

265. TRUE about hypertensive retinopathy

A. Reflects systemic hypertension
B. Causes visual blurring with an increase in blood pressure
C. Cotton wool spots represent chronic ischemia
D. Disc edema is seen only in young individuals with severe hypertension

266. Identify the sign associated with hypertensive retinopathy shown in the following image:



A. Bonnet's sign
B. Gunn's sign
C. Salu's sign
D. Amalric sign

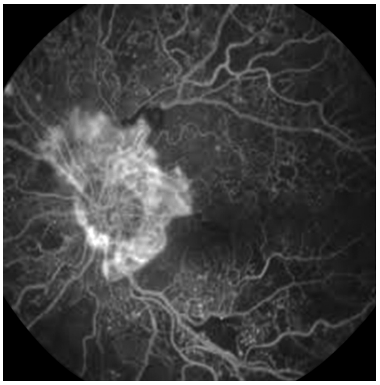
267. Seigrist streaks are seen as a part of:

A. Hypertensive retinopathy
B. Hypertensive choroidopathy
C. Hypertensive optic neuropathy
D. Hypertensive encephalopathy

261. D 262. C 263. A 264. B 265. A 266. B 267. B

268. Which of the following is the differentiating feature between grade IV hypertensive retinopathy and papilledema

A. Presence of RAPD
B. Presence of visual diminution
C. Presence of arterial attenuation of disc edema
D. Presence



269. What is the treatment for this retinopathy seen in a diabetic patient?

A. LASER photocoagulation
B. Intravitreal anti-VEGF injection
C. Both A and B
D. Follow up

270. All of the following are high-risk characteristics in PDR *except:*

A. New vessels at macula >500 microns
B. New vessels at the disc more than a one-third surface area
C. New vessels at the disc with preretinal hemorrhage
D. New vessels elsewhere more than half-disc surface

271. Features suggestive of PDR on fluorescein angiogram are:

A. Presence of leakage from vessels
B. Staining of RPE
C. Increase in arm-retina circulation time
D. Presence of intraretinal microvascular abnormality that does not leak

272. Match the following:

a. Stage I ROP	i. Demarcation ridge
b. Stage II ROP	ii. Retinal detachment
c. Stage III ROP	iii. Demarcation line
d. Stage IV ROP	iv. Demarcation ridge with extraretinal neovascularization

A. a: iii, b: i, c: iv, d: ii
B. a: i, b: ii, c: iv, d: iii
C. a: iv, b: iii, c: i, d: ii
D. a: iii, b: ii, c: i, d: iv

COMPETENCY

OP8.5

273. A patient presents with a gradual diminution of vision. Fundus examination shows a pale optic disc with clear margins and a saucerized cup and normal vasculature. What is the probable cause for this type of optic atrophy?

A. Optic neuritis
B. Papilledema
C. Pituitary tumor
D. Retinitis pigmentosa

268. C 269. C 270. A 271. A 272. A 273. C

274. The best test for color vision for acquired color blindness is:

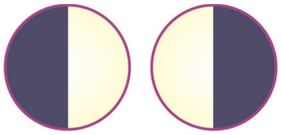
A. Ishihara
B. HRR test
C. Farnsworth Munsell test
D. Edridge Lantern test

275. Optic perineuritis is classically seen in:

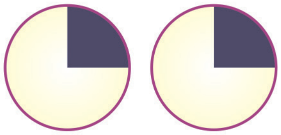
A. AION
B. Multiple sclerosis
C. Cat scratch disease
D. Syphilis

276. A patient presents with a road traffic accident. He was noted to have a hematoma in the left temporal lobe. What field defect is expected in this patient?

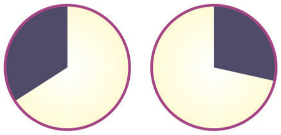
A.



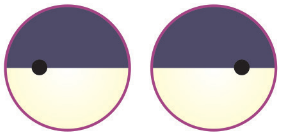
B.



C.



D.



277. Aneurysms of the posterior communicating artery are known to cause:

A. Bitemporal hemianopia
B. Junctional scotoma
C. Binasal hemianopia
D. Altitudinal hemianopia

278. Which of the following differentiates papillitis from papilledema? (multiple answers)

A. RAPD
B. Visual field defect
C. Visual loss
D. Disc edema

279. Which of the following is excluded from diagnosis for idiopathic intracranial hypertension?

A. Abducens palsy
B. Normal intracranial tension
C. Disc edema
D. Absence of other neurological features

274. C 275. D 276. B 277. C 278. A, B, C 279. B

280. A 66-year-old woman who underwent spine surgery presents with a sudden loss of vision in one eye. On examination, visual acuity is the perception of light only. The pupil is non-reactive and VEP shows decreased amplitude. The fundus is normal. Diagnosis is:

A. Anterior ischemic optic neuropathy
B. Optic neuritis
C. Posterior ischemic optic neuropathy
D. Glaucoma

COMPETENCY

OP 9.1

281. Ductions include all the following *except:*

A. Intorsion
B. Extorsion
C. Adduction
D. Levoversion

282. All of the following are features of pursuits *except:*

A. Following movements of eyes
B. Cannot be stopped once started
C. Initiated by ipsilateral occipital cortex
D. Tested by using an object moved in all directions of gaze

283. The extraocular muscles involved in levoversion are:

A. Left inferior rectus and right superior oblique
B. Left lateral rectus and right medial rectus
C. Left medial rectus and right lateral rectus
D. Left superior rectus and right inferior rectus

284. Muscles involved in dextroelevation are:

A. Right superior rectus and left inferior oblique
B. Right inferior oblique and left superior rectus
C. Right lateral rectus and left medial rectus
D. Right superior rectus and left inferior rectus

285. Match the yoke muscles:

a. Right lateral rectus	i. Left lateral rectus
b. Right superior rectus	ii. Left inferior oblique
c. Right inferior rectus	iii. Left medial rectus
d. Right medial rectus	iv. Left superior oblique

A. a: i, b: ii, c: iii, d: iv
B. a: ii, b: iii, c: i, d: iv
C. a: iv, b: ii, c: iii, d: i
D. a: iii, b: ii, c: iv, d: i

280. C 281. D 282. B 283. B 284. A 285. D

COMPETENCY

OP9.2

286. All the following are TRUE about differences between comitant and incomitant squint *except:*

A. Presence of diplopia in incomitant squint
B. Presence of past pointing in incomitant squint
C. Secondary angle of deviation is different from the primary angle of deviation
D. Presence of amblyopia in incomitant squint

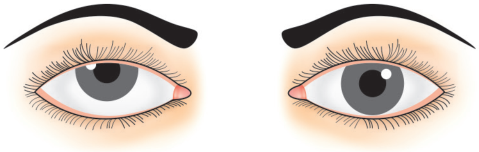
287. Which of the following is TRUE about the difference between phoria and tropia?

A. The angle of deviation in a phoria is the same at all times of the day.
B. Phoria manifests only when refractive correction is removed.
C. Tropia manifests 10 years after phoria develops
D. General debility can convert a phoria into a tropia

288. According to the Hirschberg test, what is the approximate value of 1 mm of deviation in degrees of squint?

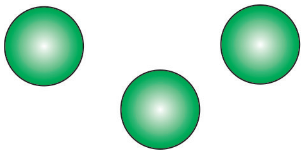
A. 7 degrees
B. 15 degrees
C. 2 degrees
D. 1 degree

289. Identify the type of squint:



A. Right hypertropia
B. Left hypotropia
C. Right hypotropia
D. Left hypertropia

290. When performing the Worth four-dot test, red-colored glass is placed in front of the right eye, and green-colored glass is placed in front of the left eye. If a person sees the following pattern, the inference is:



A. Normal BSV
B. Right suppression
C. Left suppression
D. Alternate suppression

COMPETENCY

OP9.3

291. Which of the following causes of headache is a part of primary headache syndromes?

A. Migraine
B. Papilledema
C. Uveitis
D. Refractive error

292. A 24-year-old woman presents with a unilateral headache that worsens during the menstrual period. It is associated with nausea and vomiting. Which of the following treatment options is NOT useful?

A. Sumatriptan
B. Acetazolamide
C. Ergotamine
D. Indomethacin

286. D 287. D 288. A 289. A 290 B 291. A 292. B

293. A 66-year-old woman presents with jaw claudication and bright light-associated amaurosis. She complains of right-sided temporal pain and episodes of transient loss of vision. which of the following is NOT true about this patient?

A. Associated with temporal arteritis
B. May present as ophthalmic artery occlusion
C. May present as anterior ischemic optic neuropathy
D. The headache is treated with sumatriptan

294. A 40-year-old woman presents with a unilateral headache associated with tearing of the ipsilateral eye, conjunctival congestion, miosis, and ptosis. What is the most probable diagnosis?

A. Congenital Horner syndrome
B. Acquired Horner syndrome
C. Trigeminal neuralgia
D. Migraine

COMPETENCY

OP9.4

295. The SAFE strategy is used for the elimination of which corneal blindness?

A. Trachoma
B. Onchocerciasis
C. Vitamin A deficiency
D. Infectious keratitis

296. What is the dose of oral vitamin A supplementation for infants according to CSSM protocol?

A. 50,000 IU
B. 1 lakh IU
C. 1.5 lakh IU
D. 2 lakh IU

297. Which of the following is an avoidable cause of blindness?

A. Glaucoma
B. Diabetic retinopathy
C. Cataract
D. Optic atrophy

298. The definition of blindness according to the National Programme for Control of Blindness includes all *except:*

A. Visual acuity <3/60
B. Inability to count fingers at 3 m in daylight
C. Visual field less than 10 degrees
D. Inability to appreciate primary colors

COMPETENCY

OP9.5

299. The most common site for rupture of the globe is:

A. Cornea
B. At the site of insertion of extraocular muscles
C. At the site of entry of vortex veins
D. At the site of Schlemm's canal

293. D 294. C 295. A 296. B 297. C 298. D 299. D

300. All of the following are manifestations of blunt trauma in the lens *except:*

A. Rosette cataract
B. Subluxated lens
C. Sunflower cataract
D. Vossious ring

301. A 38-year-old man met with an accident on the left side of his face following which he has severe diminution of vision in the left eye. Which of the following is a feature of traumatic optic neuropathy expected in this case?

A. Disc edema
B. RAPD
C. Reversible blindness
D. Normal visual field

302. A corneal laceration should be ideally repaired:

A. Within half hour
B. Within 6 hours
C. Within 24 hours
D. Need not be repaired as it heals by self

303. Sterile endophthalmitis is caused by which foreign body?

A. Pure gold
B. Pure silver
C. Pure copper
D. Pure iron

304. A 21-year-old female was brought with a chemical injury to both eyes. The cornea is hazy, but the iris details are partly seen. Limbal ischemia is present from 12 o'clock to 2 o'clock position. What is the grade of Roper Hall classification for chemical injury?

A. Grade I
B. Grade II
C. Grade III
D. Grade IV

305. Which of the following helps in differentiating CRAO from commotio retinae?

A. Cherry red spot
B. Decreased visual acuity
C. Whitening of retina
D. RAPD

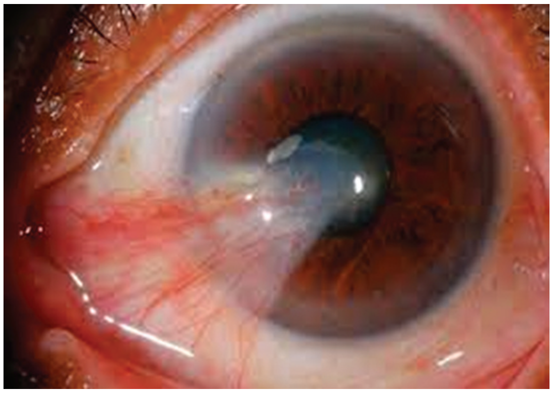
300. C 301. B 302. B 303. C 304. B 305. D

Fill in the Blanks

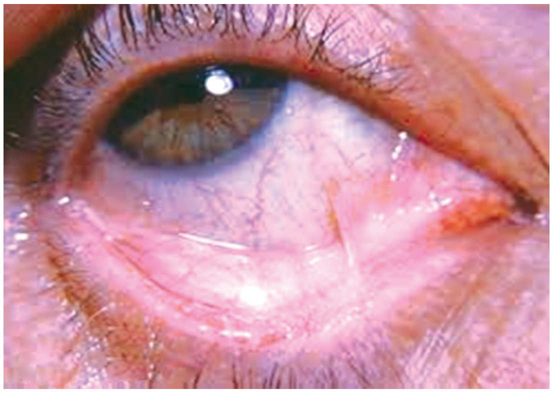
1. The process by which light energy is converted into electric energy is called _______.
2. The trichromatic theory was proposed by _______.
3. The average normal dark adaptation time is _______.
4. Silk shot retina is seen as a characteristic feature of _______ refractive error.
5. Concave lenses are used to correct _______.
6. Foster Fuch's spot is seen in _______ myopia.
7. The type of refractive error in aphakia is _______.
8. The near point is measured using the _______ rule.
9. If a patient can read the topmost letter in Snellen's chart, then his visual acuity is _______.
10. If the reflex in the pupil moves against the direction of the light shone in retinoscopy, then the refractive error is _______.
11. The instrument used for the refinement of the cylinder at the end of refraction is _______.
12. The Indian scientist who discovered excimer LASER is _______.
13. The wavelength of excimer LASER is _______.
14. _______ LASER causes photodisruption.
15. To clinically define amblyopia, the difference of visual acuity between the two eyes should be _______ Snellen lines.
16. To cause anisometropic amblyopia in myopia, the difference between two eyes should be_______ diopters.
17. A dense unilateral congenital cataract causes _______ type of amblyopia.
18. According to Listing's reduced eye, there are _______ cardinal points.
19. The nodal point lies at _______.
20. The duochrome test is based on the _______ aberration of the eye.
21. Quickert's procedure is used in correction of _______.
22. Ptosis that improves with rest is characteristic of _______.
23. In the oculosympathetic pathway, the postganglionic fibers begin from _______.
24. _______ method is used to measure the levator function.
25. When the horizontal width of the palpebral aperture is lesser than normal, it is termed as _______.
26. Marginal reflex distance-1 + margin reflex distance-2 = _______.
27. During lacrimal syringing, there is a regurgitation of saline from the same punctum. This points to an obstruction in _______.
28. Snap test is used to demonstrate _______ lid laxity.
29. _______ instrument is used to perform lacrimal probing.
30. The name of the massage technique used for treating congenital nasolacrimal duct obstruction in neonates is _______.

31. Soft stop during lacrimal probing indicates ______ obstruction.
32. ______ Sinusitis is commonly associated with orbital cellulitis.
33. Modified _________ grading is used for grading orbital inflammation.
34. Cavernous sinus thrombosis is ________ grade of orbital inflammation.
35. _________ nerve lies within the substance of the cavernous sinus.
36. Pulsatile proptosis, orbital thrill, and cork-screw conjunctival vessels are characteristic of ___________.
37. ________ sinus connects cavernous sinus to the sigmoid sinus.
38. The interleukin that plays an important role in the pathogenesis of thyroid ophthalmopathy is ________.
39. The presence of corneal exposure is _____ stage of thyroid ophthalmopathy.
40. Infrequent blinking in thyroid orbitopathy is __________ sign.
41. Idiopathic granulomatous inflammation of the orbital apex is called _________ syndrome.
42. ______ syndrome is associated with brachycephaly, pseudoproptosis, and maxillary hypoplasia.
43. Optic nerve glioma is frequently associated with ______ syndrome.
44. The most common orbital malignancy of childhood is __________.
45. The tumor that is known to 'hug' the globe without invading it is orbital _________.
46. The section of the orbit that cannot be displayed in a CT scan is ________ section.
47. The best investigative tool for studying the orbital apex is ______.
48. Tram-track calcification of the orbital tumor is seen in _______.
49. The cause of red eye that is associated with ciliary tenderness is __________.
50. When exposed to UV light, the cause of red eye is ________.
51. Ciliary congestion is seen in _________.
52. Ciliary tenderness is elicited by pressing at ____ mm from the limbus.
53. _____ congestion does not blanch following application of topical phenylephrine.
54. The condition that is associated with the appearance of both papillae and follicles is _________.
55. Chemical conjunctivitis in neonates is caused by the application of _______.
56. Hyperacute conjunctivitis is caused by _______.
57. True membranous conjunctivitis is caused typically by __________.
58. A noninfective cause of membranous conjunctivitis is __________.
59. The antibiotic of choice in angular conjunctivitis is __________.
60. The type of conjunctivitis seen in association of COVID-19 is ___________.
61. Subconjunctival scarring associated with trachoma is called __________.
62. McCallan classifies trachoma into ______ stages.
63. Neonatal inclusion conjunctivitis is caused _____ serotypes of *Chlamydia*.
64. Shield ulcer is treated with ________ drug.
65. ________ conjunctivitis is seen as a hypersensitivity to tubercular protein.
66. Dennie Morgan fold is seen in association with ______ conjunctivitis.

67. The lesion shown in the image is _________.

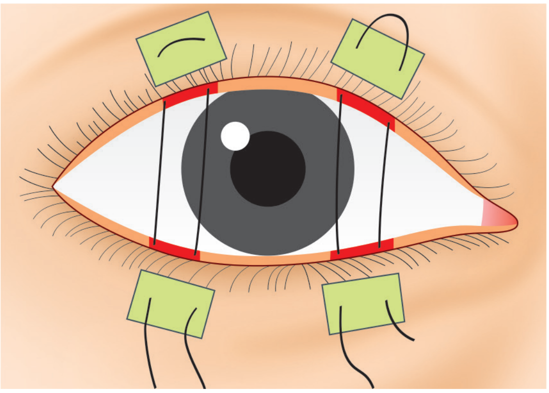


68. The standard of care procedure for pterygium excision is ________.
69. The concentration of mitomycin-C used in pterygium excision is _________.
70. Symblepharon is the adhesions formed between __________ and ______ conjunctiva.
71. Untreated chemical injury can lead to the formation of adhesions between the upper and lower lids. This is called _________.
72. The lesion shown in the image is __________.



73. Ophthalmia nodosa is caused by __________.
74. A Rust ring in the cornea is formed by _______ foreign body in the cornea.
75. The instrument used to perform double eversion is ________.
76. ________ is an example for preservative used in ocular solutions.
77. The form of atropine used for refraction in infants is _________.
78. A method of decreasing systemic absorption through the nasolacrimal duct is ______.
79. The lateral one-third of the upper palpebral conjunctiva drains into ________ lymph nodes.
80. _______ bacterium thrives well in dry conjunctiva that is associated with vitamin A deficiency.
81. The innermost layer of conjunctiva is called __________.
82. The number of layers in the cornea is ______.

83. The fifth layer of the cornea is named after ___________.
84. _______ test uses fluorescein dye to diagnose corneal microperforation.
85. Hypopyon corneal ulcer is caused by ___________.
86. Painless corneal ulceration is seen in_________.
87. The most common organism implicated in keratitis associated with a blocked nasolacrimal duct is __________.
88. The preferred treatment for herpetic keratitis in the pre-antiviral era was __________.
89. Wesley's immune ring is characteristically seen in _________ keratitis.
90. A posterior corneal defect is seen in the ______ anomaly.
91. The average number of endothelial cells per mm^3 in a 25-year-old man is ______.
92. The investigation used to study the endothelium is called _________.
93. The type of dry eye associated with Sjögren syndrome is _____________.
94. The normal value of Schirmer test at the end of 5 minutes is ___________.
95. ___________ drug used for treating acne causes dry eye.
96. Opacification of the cornea that allows visualization of the details of the iris is called ___________ grade of opacity.
97. River blindness is caused by ______________.
98. The endothelial dystrophy that manifests in the 5th decade is __________.
99. Keratoplasty performed by maintaining the integrity of the globe is called ___________ keratoplasty.
100. Superficial corneal scar can be treated by _____________.
101. Khodadaust line is a sign of _____ rejection in a corneal graft.
102. The most common indication for tarsorrhaphy is ______________.
103. The type of tarsorrhaphy shown in the image is _______________.



104. The corneal foreign body that can cause formation of a rust ring is made up of ____________.
105. The drug used to anesthetize the ocular surface before attempting to remove a foreign body is __________.
106. The time before which the cornea should be retrieved from the deceased individual is _______.
107. The presence of marked deep folds in the Descemet's folds indicates ____ grade of a corneal button.

108. The instrument used to remove a corneal button from the enucleated eye is ________.
109. A corneal button can be stored in K-sol for ________ days.
110. The investigation that is used to study the surface of the cornea is _____________.
111. Megalocornea is diagnosed if the horizontal diameter of the cornea is more than ______.
112. The corneal degeneration associated with hypercalcemia is ___________.
113. The most common systemic association of episcleritis is _______________.
114. The infectious etiology that can simulate episcleritis is ______________.
115. The first line of drug for the treatment of episcleritis is ___________.
116. Scleromalacia perforans is associated with ___________.
117. The diagnostic sign for posterior scleritis on B-scan is _____________.
118. A ciliary staphyloma is usually due to _______________.
119. "Headlight in the fog" appearance is typically described for __________ associated uveitis.
120. Juvenile idiopathic uveitis is associated with Still's triad. The uveitis that is seen is usually ___________.
121. ___________ differentiates endophthalmitis from panophthalmitis.
122. The most used intravitreal antibiotics for postoperative endophthalmitis are ______ and ___________.
123. The presence of 14 cells per mm^2 of the anterior chamber denotes activity score ________.
124. The most common cause of nongranulomatous anterior uveitis is ____________.
125. Uveitis lasting for more than 3 months is termed ___________.
126. The corneal manifestation of chronic uveitis is ___________.
127. A sightless, shapeless, eye with no vision is called ___________.
128. Uveoparotid fever is also called _____________.
129. The HLA associated with ankylosing spondylosis is ___________.
130. Cold hypopyon is typically described in _________ syndrome.
131. Inverse hypopyon is seen in ____________.
132. A non-traumatic cause for hyphema is ____________.
133. Total hyphema is also known as ___________.
134. Grade II of the angle of the anterior chamber is defined when the structures are seen up to ____________.
135. _________ grading is used to measure the angle of the anterior chamber and probability of angle-closure attack.
136. The blood-aqueous barrier is formed between the ________ cells of the ciliary processes.
137. The anterior-most structure seen in gonioscopy is __________.
138. _______ are seen during gonioscopy in 100-day glaucoma.
139. A widened ciliary body band is characteristically seen in __________ glaucoma.
140. Splinter hemorrhages at the disc margin seen in progressive glaucoma are called ________ hemorrhages.
141. The concentration of mannitol used in reducing IOP in acute angle-closure glaucoma is ___________.
142. The LASER used for performing peripheral iridotomy is _________.

143. The instrument used in performing goniotomy in congenital glaucoma is __________.
144. Brinzolamide belongs to the _______ class of antiglaucoma drugs.
145. _________ drug has neuroprotective action.
146. The best investigation of choice in a child with chronic uveitis and joint inflammation is _________.
147. A child is brought with bilateral nystagmus and fundus examination reveals a solitary macular healed choroiditic patch. The best possible investigation is __________.
148. The test that measures the interferon levels in tuberculosis is __________.
149. The cycloplegic of choice for anterior uveitis is ____________.
150. The antiglaucoma medication that is contraindicated in hypertensive uveitis is _________.
151. _______ is an IL-2 antagonist.
152. ___________ is described as white-eyed uveitis.
153. The potential side effect of using topical steroids is ___________
154. The thinnest part of the human crystalline lens is _________.
155. The lens is placed in the ___________ fossa behind the iris.
156. The lens develops from the __________ (germinal layer).
157. Iris shadow is seen in __________ cataract.
158. Cataracta nigra is _____ grade of nuclear cataract.
159. The cataract associated with blunt trauma is ________.
160. The formula used to calculate the power of the IOL for cataract surgery is _______.
161. The only cataract that is reversible in the initial stages is the one associated with ___________.
162. Tremulousness of lens is suggestive of ____________ of lens.
163. ________ is used to grade various types of cataracts.
164. A 73-year-old patient was being evaluated for surgery. He was on Tamsulosin for bladder symptoms. A poorly dilating pupil is suggestive of ____________..
165. The length of incision in SICS is _________.
166. The method by which the nucleus is made mobile in the capsular bag by using fluid is called __________.
167. An expected complication in a posterior polar cataract during hydrodissection is _____________.
168. A small capsulorhexis in a brown cataract predisposed to ___________ complication during nucleus delivery.
169. UGH syndrome is seen with the use of _______ IOLs.
170. Following consent for cataract surgery, the patient is given an injection of anesthetic near the eyeball. It is called _____________.
171. The contents used for peribulbar anesthesia include lignocaine, adrenaline, and ___________.
172. Oculocardiac reflex is also called ____________ reflex.
173. A patient with a macular hole and a cataract opted for cataract surgery. _______ test will help you ascertain poor macular function
174. Hollenhorst plaques are made up of _______________.
175. A cause of painful CRAO is ___________.
176. A vessel arising from the ciliary circulation that supplies macula in a variable number of individuals is ______.

177. The indication of LASER photocoagulation in a CRVO is the presence of ___ clock hours of neovascularization.
178. The LASER used for panretinal photocoagulation is __________.
179. ________ is a complication of panretinal photocoagulation.
180. When the laser beams are used to blanch the microaneurysms, it is termed __________ laser.
181. The distance between the foveola and the temporal disc margin is ________mm.
182. The magnification with a direct ophthalmoscope is ________.
183. The size of the foveal avascular zone is ______.
184. A patient with severe NPDR must be reviewed every _______ months.
185. Presence of IRMA classified the retinopathy as _________.
186. _______ is the most used anti-VEGF drug in treating vasculoproliferative disorders of the retina.
187. Normal vasculogenesis of the retina is complete by ______ weeks of gestation.
188. Oculodigital reflex is seen in __________.
189. In the optic chiasm, ___________ fibers cross to the opposite side.
190. Homonymous hemianopia is seen lesions posterior to ___________.
191. __________ optic neuropathy is associated with a normal appearing disc in early stages.
192. Foster Kennedy syndrome is typically described in ____________.
193. Pallid disc edema is typically described in __________.
194. Retina has ________ layers on microscopy.
195. Vitreous opacities that form due to deposition of calcium deposition in the vitreous are called _________.
196. Primary vitreous is ___________ in origin.
197. Bilateral conjugate movement of eyes is termed __________.
198. The muscles responsible for dextroversion right ____________ and left ____________.
199. The muscles that do not arise from the annulus of Zinn are __________ and __________.
200. Latent squint is also known as ________.
201. The primary angle of deviation and secondary angle of deviation is equal in _________ squint.
202. __________ squint are corrected with muscle transpositions.
203. In ________ squint, forced duction test is positive.
204. While performing Worth's four-dot test, a patient seen only two red dots. This indicates ________.
205. The most common type of refractive error that causes headache is _______________.
206. An example for trigeminal neuralgia is ___________.
207. The definition of blindness according to NPCB is visual acuity less than ___________.
208. Most common cause of avoidable blindness is ___________.
209. According to CSSM protocol, a 9-month-old child should receive ________ amount of oral vitamin A.
210. Siderosis bulbi is caused by _________ intraocular foreign body.
211. The angle anomaly associated with blunt ocular trauma is __________.
212. D-shaped pupil is seen in _________.

213. A patient came with an alkali injury to his right eye. On examination, there is limbal whitening extending from 3 o'clock to 6 o'clock position. The grade of chemical injury is __________.
214. The retinal manifestation of blunt trauma that shows a cherry-red spot is __________.
215. Physiological nystagmus is usually ______ type.
216. The horizontal gaze center is in the ____________.
217. An example for unilateral nystagmus is _____________.

ANSWERS

1. Phototransduction
2. Young and Helmholtz
3. 20 minutes
4. Hypermetropia
5. Myopia
6. Degenerative/pathological
7. Hypermetropia
8. Royal Airforce/RAF
9. 6/60
10. Myopia >1D.
11. Jackson's cross-cylinder/astigmatic fan
12. R Srinivasan
13. 193 nm
14. Femtosecond
15. 2
16. 4
17. Stimulation deprivation
18. 4
19. Posterior capsule of the lens
20. Chromatic
21. Entropion
22. Myasthenia gravis
23. Superior cervical ganglion
24. Berke
25. Blepharophimosis
26. Palpebral fissure height
27. Canaliculus
28. Horizontal
29. Bowman's probe
30. Crigler massage
31. Canalicular
32. Ethmoidal
33. Chandler's
34. V
35. Abducens
36. Caroticocavernous fistula
37. Superior petrosal
38. 1β
39. V
40. Stellwag
41. Tolosa–Hunt
42. Crouzon
43. Neurofibromatosis I
44. Rhabdomyosarcoma
45. Lymphoma
46. Sagittal
47. MRI
48. Optic nerve sheath meningioma
49. Anterior uveitis
50. UV keratopathy
51. Anterior uveitis/acute congestive glaucoma
52. 2
53. Ciliary
54. Trachoma
55. Silver nitrate
56. *Gonococcus*
57. *Corynebacterium diphtheriae*
58. Ligneous conjunctivitis
59. Tetracycline
60. Follicular conjunctivitis
61. Artl's line
62. 4
63. D-K
64. Corticosteroid
65. Phlyctenular
66. Atopic
67. Pterygium
68. Conjunctival autograft

69. 0.02%
70. Bulbar and palpebral
71. Ankyloblepharon
72. Symblepharon
73. Caterpillar hair
74. Metallic/iron
75. Desmarre's retractor
76. Benzalkonium chloride
77. Topical ointment
78. Punctal occlusion
79. Preauricular
80. Corynebacterium xerosis
81. Fibrous layer
82. 6
83. Harminder Singh Dua
84. Seidel
85. *Pneumococcus*
86. Neurotrophic keratitis
87. *Pneumococcus*
88. Debridement
89. Fungal
90. Peter's
91. 4000
92. Specular microscopy
93. Aqueous deficiency
94. 15 mm
95. Retinoic acid
96. Nebula
97. Onchocerca volvulus
98. Fuch's
99. Tectonic
100. Phototherapeutic keratectomy
101. Epithelial
102. Facial palsy
103. Paramedian tarsorrhaphy
104. Iron
105. Proparacaine
106. 6 hours
107. Grade V
108. Trephine
109. 7–10
110. Corneal topography
111. 11.7 mm
112. Band-shaped keratopathy
113. Rheumatoid arthritis
114. Conjunctival ulcer
115. NSAIDs
116. Rheumatoid arthritis
117. T-sign
118. Absolute glaucoma
119. Toxoplasmosis
120. Chronic granulomatous
121. Proptosis/chemosis/restricted ocular movements/absence of perception of light
122. Vancomycin, ceftazidime
123. 2
124. HLA B27 uveitis
125. Chronic
126. Band-shaped keratopathy
127. Phthisis bulbi
128. Heerfordt syndrome
129. B27
130. Behcet
131. Silicone oil
132. Hemophilia
133. 8-ball hyphema
134. Trabecular meshwork
135. Shaffer's
136. Non-pigmented epithelial
137. Schwalbe's line
138. New vessels
139. Angle-recession
140. Drance
141. 20%
142. Nd-YAG
143. Barkan's knife
144. Carbonic anhydrase inhibitors
145. Brimonidine/betaxolol
146. ANA
147. IgM toxoplasmosis
148. Quantiferon gamma
149. Atropine
150. Prostaglandin analogs
151. Daclizumab
152. JIA associated uveitis
153. Ocular hypertension or glaucoma
154. Posterior capsule
155. Patellar

156. Surface ectoderm
157. Immature
158. V
159. Rosette
160. SRK/Sanders Retklauff and Krauff
161. Galactosemia
162. Subluxation/weak zonules
163. Lens opacification classification system
164. Floppy iris syndrome
165. 6 mm
166. Hydrodissection
167. Posterior capsular tear
168. Zonular dialysis
169. Anterior chamber
170. Retrobulbar anesthesia or peribulbar anesthesia
171. Hyaluronidase
172. Ashner's reflex
173. Photostress recovery test
174. Cholesterol
175. Giant cell arteritis
176. Cilioretinal artery
177. 2
178. Argon-green
179. Uveal effusion/optic atrophy
180. Focal
181. 3
182. 10x
183. 500 µm
184. 3
185. Severe/very severe NPDR
186. Bevacizumab
187. 40
188. Leber congenital amaurosis
189. Nasal
190. Optic chiasm
191. Traumatic
192. Olfactory groove tumor
193. Arteritic anterior ischemic optic neuropathy
194. Ten
195. Asteroid hyalosis
196. Mesenchymal
197. Version
198. Right lateral rectus, left medial rectus
199. Superior oblique and inferior oblique
200. Phoria
201. Comitant
202. Paralytic
203. Restrictive
204. Left suppression
205. Hypermetropia
206. Cluster headache
207. 6/60
208. Cataract
209. 1 lakh
210. Iron
211. Angle recession
212. Iridodialysis
213. III
214. Berlin's edema
215. Jerk
216. Parapontine reticular formation
217. Internuclear ophthalmoplegia

Competency Based Questions and Answers in
Ophthalmology

for Third MBBS
Professional
Examination

The main objective of this book is to introduce to the undergraduate students the subject of ophthalmology in a simplified manner in the form of questions and answers. The new CBME curriculum brings with it a finely structured method of learning both theory and practical aspects of ophthalmology. Also, the upcoming pattern of evaluation is another overwhelming change that a student must gear up for NEXT. Keeping these two new developments in mind, the questions are framed using Bloom's taxonomy verbs and the answers are written in a structured format using bulleted points to ease learning and memorizing.

Salient Features

- Questions and MCQs have been framed as per the CBME guidelines
- Contents have been arranged as per the competencies set under the CBME guidelines, including both theory and practical components
- Conventional long essays, short essays, short answers, multiple choice questions of various types, and ‘fill in the blanks’ have been included for broader experience of the student
- Many image-based (both clinical and investigation) questions have been added
- All answers are heavily supported by numerous diagrams and flowcharts
- Some topics that were excluded under a separate heading for overall understanding of ophthalmology

Madhurima K Nayak MS, DNB is currently Consultant Ophthalmologist, Yenepoya Speciality Hospital, Mangaluru, Karnataka. She did her MBBS and MS in ophthalmology from Kasturba Medical College, Manipal University, Mangaluru, in 2011 and 2014 respectively. She secured DNB from the National Board of Examinations in Medical Sciences in 2015. She worked in the same institution as a senior resident for 2 years during which she was actively involved in academic activities. She has a special interest in neuro-ophthalmology and uveitis. She has to her credit around 30 papers published in national and international journals. She has co-authored *Competency Based Questions and Answers in Physiology*.

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CBS Publishers & Distributors Pvt Ltd
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